ARCHIVES OF OPHTHALMOLOGY

VOLUME 37 (old series Volume 94) JANUARY 1947 NUMBER 1

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OCULAR SURGERY

Random Observations

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AS AN ocular surgeon and teacher for more than twenty years, one naturally forms certain judgments as to surgical methods and procedures. These are offered herewith in the hope that they may be of benefit to others. Although it is realized that many may be an old story, others may be of interest.

ANESTHESIA

No excuse exists for poor anesthesia. A fearful and uncooperative patient makes for a poor surgical performance.

Preoperative sedation is indicated for all patients. Morphine should not be given before intraocular operations, since it may lead to vomiting. However, its use is quite satisfactory for extraocular surgical procedures.

Cocaine hydrochloride is probably the best of the local anesthetics for instillation. The lids should be kept closed, since cocaine disturbs the corneal epithelium to a less extent under this condition. Also, cocaine should not be instilled for more than ten or twelve minutes; longer periods tend to soften the cornea. Subconjunctival injection of a weak solution of cocaine or procaine enhances deep anesthesia. Since tetracaine hydrochloride, 0.5 per cent, does not act as a mydriatic, it is substituted for cocaine in operations for glaucoma.

A retrobulbar injection of 2 per cent procaine hydrochloride with epinephrine hydrochloride, 1 : 20,000, results in deep anesthesia of all intraocular structures. This injection should be given into the muscle cone as near the globe as possible in order to reach the ciliary nerves.

AKINESIA

Paralysis of the muscles of lid closure is indicated in any operation in which the globe is to be opened. This is especially true in cataract extraction, in which the incision is large and squeezing may result in loss of vitreous. It is believed that the injection of 2 per cent procaine hydrochloride with epinephrine hydrochloride, 1 : 20,000, over the condyloid process of the mandible is the procedure of choice.

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Read at a meeting of the New England Ophthalmological Society, Boston, Dec. 18, 1945.

PREPARATION FOR INTRAOCULAR OPERATIONS

Epinephrine hydrochloride, 1:1,000, instilled just before an intraocular operation induces hemostasis and lessens hemorrhage materially.

It is impossible to sterilize the cornea and conjunctiva, but painting the lid margins, lids and surrounding skin with one-half strength tincture of iodine lessens the chances of infection.

Prior to cataract extraction with peripheral iridotomy or iridectomy the pupil is dilated with homatropine and paredrine hydrobromide ophthalmic.[1]

CATARACT EXTRACTION

The injection of typhoid H antigen vaccine intravenously, in a dose of 10,000,000 to 15,000,000 bacilli, two days before operation and repetition, with a dose of 15,000,000 to 20,000,000, on the day prior to operation are routine, and the procedure is believed to lessen postoperative iridocyclitis. Also, in an attempt to reduce infection at operation, penicillin ointment is instilled into the conjunctival sac every two hours for two days.

In modern surgical procedures, it goes without saying that sutures are to be used. Certainly, the corneoscleral type, placed before the incision is made, is desirable. The advantages of such sutures are several: rapid closure of the wound if loss of vitreous is imminent or has occurred, early reformation of the anterior chamber, infrequency of wound separation, rarity of prolapse of the iris, infrequency of postoperative glaucoma, lessening of astigmatism, prevention of corneal eversion and increased activity of the patient during convalescence.

A slightly modified Stallard suture is satisfactory. A knot is made approximately 3 cm. from the end of a 000000 silk suture, which is armed with an atraumatic needle. At 12 o'clock, the suture is passed through the anterior layers of the cornea very close to and parallel with the limbus and then pulled through until the knot rests against the corneal surface. Then directly above and at a distance of about 2 mm., the suture is passed through the conjunctiva and the superficial layers of the sclera in a direction parallel with the corneal suture. This is really just a mattress suture across the limbus. The loops are spread to make way for the incision, iridotomy and delivery of the lens. By pulling on the end of the suture opposite the knot, the wound may be instantly closed at any time.

Simple delicate instruments are always desirable. Use of complicated speculums, locked fixation forceps, and the like, is not advisable, since in case of accident one must be able to get out rapidly.

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1. Paredrine hydrobromide ophthalmic is a 1 per cent solution of parahydroxy-α-methylphenylethylamine hydrobromide in distilled water, made isotonic with 2 per cent boric acid and preserved with merthiolate, 1:50,000.

The keratome-scissors incision is much more easily made and is safer than the full Graefe knife incision. Admittedly, it is not so spectacular and may hurt one's pride a bit; but, after all, the patient should receive first consideration. This incision is not such a mental hazard for the beginner or the occasional operator. It is almost impossible to make a poor incision with the keratome and scissors; fixation need not be so secure and if lost may be easily regained; the incision may be made more slowly; aqueous is not lost until the keratome incision is complete; there is no prolapse of iris during the process, and, finally, the incision seems to heal more rapidly and firmly. When the incision is completed with scissors, the outside blade is to be laid against the sclera before cutting, since thus the incision is back of the limbus and sutures are more easily placed.

In the presence of a very shallow anterior chamber a short Graefe knife incision may be made more easily. Or, better still, a rather long scratch incision is made 1 mm. back of and parallel with the limbus and then completed with scissors.

A peripheral iridotomy is desirable, even though delivery of the lens is somewhat more difficult. This handicap is overcome if the assistant exerts counterpressure above during delivery of the lens. Prolapse of the iris is not more common with peripheral iridotomy if corneoscleral sutures are used. The eye has a better appearance, and perhaps the vision is somewhat more acute.

Intracapsular extraction of the lens is advisable. A large bite of capsule is preferable to a small one. During this stage of the operation the speculum should be lifted away from the globe by the assistant; this removes all pressure from the globe and often actually creates suction, as may be seen by the corneal dimpling. When one is delivering the lens through a round pupil, countepressure exerted just above the upper limbus by the assistant aids in forcing the lens through the opening. Such pressure is released as soon as the lens presents in the wound.

Immediately after extraction of the lens the mattress suture is tied, and four ordinary sutures are placed across the incision; they pass through the tough limbal tissues, on one side, and through episclera and conjunctiva, on the other.

Air injected into the anterior chamber keeps open the angle, thus discouraging formation of synechias; also, it helps to prevent prolapse of the iris during the first three or four days of healing. Care should be exercised to fill only about two thirds of the chamber with air and to keep the air in front of the iris. If air passes through the pupil into the posterior chamber it may cause prolapse of the iris or acute glaucoma by forcing the iris against the chamber angle.

A 1 per cent solution of physostigmine installed after the operation is of assistance in keeping the chamber angle free, in preventing prolapse of the iris and in keeping the air in the anterior chamber.

One eye only need have a dressing. The patient may be allowed to sit in a chair or lie in bed, as desired.

If an extracapsular extraction is made, capsule forceps are preferable to a cystotome. The [illegible] removes part of the capsule and lens epithelium; consequently [illegible]eration is lessened and after-cataract is infrequent.

[illegible]TER-CATARACT

If the membrane is old and tough, dilaceration with two knife needles may be indicated. Or one may make a small keratome incision, introduce a sharp iris hook, pull the membrane out and cut it off.

CATARACT AND PRIMARY GLAUCOMA

Opinions differ widely as to the surgical procedure in cases of such a complication. Some surgeons prefer to do a preliminary iridectomy; others do a cyclodialysis, but most operators perform an external filtering operation before cataract extraction. In my experience, if an external filtering operation is done first and, at a later date, is followed with the usual cataract incision through the bleb, the intraocular pressure often rises as a result of scar formation within the former filtering cicatrix. One may, however, make a corneal incision below the bleb, or a lateral incision which does not extend into the filtering area.

Other surgeons make a cataract extraction and at the same time perform a complete iridectomy or an iris inclusion. If the cataract extraction is performed first, it is difficult to do at a later date an external filtering operation because of the scar.

More satisfactory, it seems, is a combined anterior sclerectomy (Lagrange) and cataract extraction. An injection of 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride, 1 : 20,000, is made between Tenon's capsule and the sclera above the upper portion of the limbus. Such an injection makes dissection easier and insures a thicker flap. The flap is dissected down as usual for the Lagrange operation, and a keratome incision is made well back of the limbus. Then, with a Holth punch, a bit of sclera is removed. As the next step, a peripheral basal iridectomy is done. After this, the incision is enlarged with scissors and the lens is extracted intracapsularly in the usual manner. Conjunctival sutures complete the operation.

Objection has been made to the combined anterior sclerectomy and cataract extraction because of the slow reformation of the anterior chamber and perhaps the exaggerated formation of anterior synechias. In my experience, the anterior chamber reforms almost as rapidly as

after cataract extraction. Often anterior synechias are already present and the angle is closed before operation; otherwise, a filtering operation would not be indicated.

OPERATIONS FOR GLAUCOMA

No operation is always successful in the treatment of glaucoma, even though a complete study of the case is made, the diagnosis is correct and the operation is apparently well done.

Acute Narrow Angle Glaucoma (acute congestive glaucoma).—In the early stages, i. e., within the first two or three days, or if the tension returns to normal with use of miotics, a basal iridectomy is indicated. The incision should not be made with a keratome, for it is almost always anterior to the chamber angle. An incision with a Graefe knife is better, but the anterior chamber is usually emptied too rapidly and the iris tends to prolapse before an iridectomy can be done; this is not especially unsatisfactory if one wishes to do a complete iridectomy. But if a peripheral basal iridectomy is desired the iris must be replaced first, oftentimes a difficult, or even impossible, procedure. The best technic for iridectomy is to enter the anterior chamber through a scratch incision 1.5 mm. back of and parallel with the limbus; the aqueous may be emptied slowly, and a peripheral basal iridectomy is a simple procedure.

Chronic Wide or Narrow Angle Glaucoma (glaucoma simplex).—Cyclodialysis is good practice in cases of chronic wide or narrow angle glaucoma in which the tension is not too greatly elevated, in cases with small visual fields or as a second operation. It is also indicated in cases of postoperative glaucoma following cataract extraction.

Before cyclodialysis is performed, the chamber angle is studied with the gonioscope to determine the position of dense synechias and larger vessels, both of which are to be avoided at operation if possible.

After the conjunctiva is opened, a scleral fixation suture is placed just ahead of the area chosen for the incision. A 0000 twisted silk suture on an atraumatic needle is passed through the anterior layers of the sclera; the two ends are cut short and then grasped close to the sclera with a small hemostat. This gives excellent fixation; in addition, the wound may be made to gape for introduction of the iris repositor.

The iris repositor should be passed through the attachment of the ciliary body with pushing rather, than with sweeping, movements. One should not go far into the anterior chamber with the spatula, otherwise, the corneal endothelium and Descemet's membrane may suffer undue damage. An absorbent applicator placed at the incision assists in the prevention of hemorrhage into the anterior chamber.

It seems that almost one-half the attachment of the ciliary body should be torn away. Probably the operation has fallen into disrepute because of too little dialysis.

External Filtering Operations.—In the Lagrange or Elliott operation, injection of fluid under Tenon's capsule helps to obtain a thick flap. A thick flap, which should include both conjunctiva and Tenon's capsule, protects against late infection through the area of the bleb.

CONVERGENT STRABISMUS

Indications for operation are based on the ocular condition, and not on the age of the patient. It is not wise to perform operation for squint until an attempt has been made to correct it with glasses; to obtain as nearly normal visual acuity as possible, and to develop (1) single binocular vision, (2) normal retinal correspondence and (3) fusion, with some amplitude at the objective angle. It is understood that this cannot always be accomplished—if not, an operation may be performed for cosmetic reasons.

General anesthesia is best for such operations, and pentothal sodium given intravenously can be recommended in operations on children over 5 or 6 years of age.

It seems wise to do a two stage operation in cases of convergent squint, for with this method there are few overcorrections. Undercorrection is expected after the first operation. In most cases of convergent squint of over 15 degrees a recession of 5 mm. is made on one internal rectus muscle. After three or more months the lateral rectus is shortened or shortened and advanced, depending on the amount of squint remaining. One never knows the exact amount of recession or resection to make; only great experience is of value; therefore the two stage operation is advisable.

My greatest failures have been with patients who are older and have very poor vision in one eye; those with severe spasm of the inferior oblique, those with abnormal retinal correspondence and, especially, those with eccentric fixation.

In a recession a muscle clamp is never used, since 2 to 3 mm. of muscle is lost. Only a squint hook is used, and the sutures are placed as close to the insertion of the muscle as possible. The muscle sheath is always kept intact.

In cases of resection or of resection and advancement the muscle is attached to the sclera with three sutures. This allows the patient complete use of the eyes soon after operation.

The use of surgical gut U. S. P. (catgut) sutures is advised for both muscle and conjunctiva. Removal of the sutures is then unnecessary. After the operation a thin dressing and an ice cap or ice glove for forty-eight hours reduce the postoperative reaction.

The eyes are allowed to remain open after the ice cap is removed. In cases of advancement in which tension is exerted on the sutures, pin-

hole glasses may be prescribed for a few days in order to reduce ocular movements.

TUMORS OF THE ORBIT

After having tried the Krönlein and the brow incision, I have for the past several years used to advantage the following approach to orbital tumors: A wide lateral canthotomy, extending about 1 cm. back of the bony orbital margin, is made, and the incision is extended upward or downward in the conjunctival fornix. All tumors except those nasal to the muscle cone may be reached by this incision.

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MALIGNANT MELANOMA OF LIMBUS AND SPONTANEOUS CYST OF PIGMENTED LAYER OF IRIS IN SAME SECTOR

Contribution to Histogenesis of Tumors

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A CASE is reported in which a malignant melanoma of the limbus and a cyst on the posterior surface of the iris occurred in the same sector of the eye.

REPORT OF A CASE

A woman aged 33 had always had defective vision in both eyes, and her eyes had been examined annually. The routine test in 1944 had shown no alteration from the previous year, but in 1945 her optician noticed for the first time a dark spot at 9 o'clock on the limbus of the right eye; a month later this was found to have increased rapidly in size. No history could be obtained of injury, inflammation or a mole prior to her last refraction.

Examination.—Right Eye: With a correction of + 3.75 D. cyl., axis 180 vision was 6/9. A sausage-shaped, slightly elevated melanotic tumor overlay and was concentric with the limbus from 8 to 11 o'clock. It was 5 mm. long, 2 mm. wide and 1 mm. thick (fig. 1).

Minute tongues of melanotic infiltrations were visible temporally, and pigment spots extended along the small conjunctival and subconjunctival vessels for 1 mm. No such infiltration was visible in the cornea, but small, stellate pigment spots (resembling those composing a congenital pupillary membrane) were scattered in the corneal epithelium 3 or 4 mm. around the tumor. The lens was studded with half a dozen small, punctate anterior capsular cataracts, with reduplication. No sign of posterior synechias or of a pupillary membrane was seen with the biomicroscope, and the fundus was normal except for one spot of pigment in the temporal periphery.

Left Eye: With a correction of —1.0 D. vision was 6/9. This eye also displayed multiple anterior capsular cataracts, with a tendency to reduplication. One of these opacities projected above the surface like an iceberg. Synechias, suggesting old iritis, were present, but no threads of pupillary membrane were seen.

Operation.—The tumor was separated from the cornea and episclera with Tooke's knife and scissors in an attempt to remove it with a margin of healthy tissue on all sides. The raw area was covered with a conjunctival flap from the outer side. The eye recovered rapidly and was "white" in a week.

The histologic investigation was performed at the Tennent Institute for Ophthalmology, University of Glasgow (Prof. W. J. B. Riddell).

Biopsy of Tumor (fig. 2).—The normal squamous epithelium, with goblet cells and normal subconjunctival tissue, thinned gradually as it approached the edge of the tumor. Even here, however, the basal epithelial cells were pigmented.

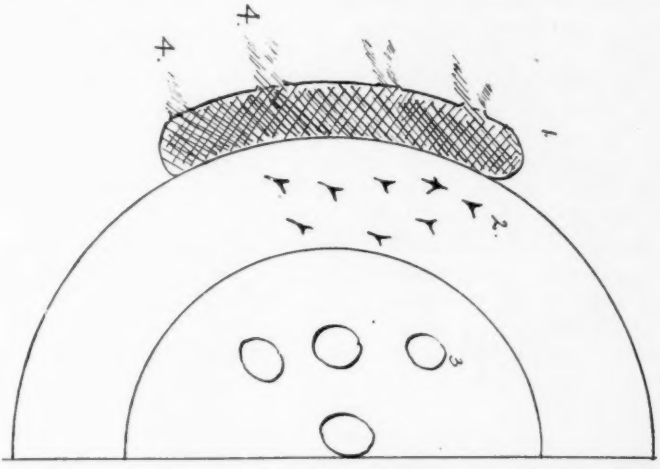

Fig. 1.—Schematic diagram of limbal melanoma. *1* indicates site of limbal tumor; *2*, corneal pigment; *3*, punctate cataract, and *4*, conjunctival infiltration.

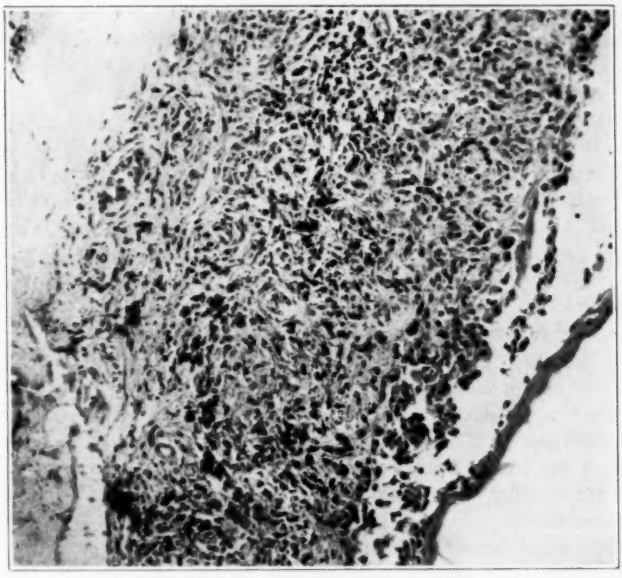

Fig. 2.—Limbal melanoma; hematoxylin and eosin, × approximately 150.

The intraepithelial islets consisted of cells of varying degrees of pigmentation, located in vacuoles with a loose, unpigmented subepithelial tissue. Even this area showed evidence of malignancy. Around these "cysts," where it overlay the

tumor proper, the epithelium thinned to one or two layers and normal subepithelial connective tissue was absent.

The tumor itself consisted of polymorphic nuclei, displaying great variations in shape, size and intensity of staining. Round, polyhedral, spindle-shaped and giant nuclei, five times the average size, were all present. There were few mitoses, and the nucleolus was as a rule indistinct. Coffee brown pigment was largely distributed in all parts of the tumor. Van Gieson's stain revealed little connective tissue, and specific staining showed a low reticulum content. Vascularization was adequate, and necrosis and inflammatory reaction were absent.

While no sharp line of demarcation was present between the tumor and the underlying normal sclera, there was no evidence of infiltration of the deeper layers in the sections we examined.

Enucleation and Examination of Excised Eye.—As the tumor cells were of an immature and malignant type, enucleation of the eye was obviously indicated. Care was taken during this operation to excise the conjunctiva widely around the site of the tumor.

Examination with the Slit Lamp: After fixation in formaldehyde-saline solution, the back of the globe was removed by a postequatorial section to permit examination with the slit lamp of the entire ciliary body from behind. The view obtained is well shown in figure 3 (painted by Gabriel Donald). The lens was in situ; zonular fibers were traceable, and Hannover's canal was visible. The anterior opacities were not visible through the opaque lens.

No abnormality of the uvea could be detected except for the small, flat, sharply demarcated tumor shown in Donald's picture, which was located at the ciliary side of the iris root and underlay approximately the center of the site of the episcleral tumor. As is also shown in the picture, around this relatively flat tumor were a number of very small, brown, grapelike bodies on the crests and in the valleys of the ciliary processes. No sign of such changes could be found elsewhere with the slit lamp.

Histologic Examination: The eye was embedded in pyroxylin, and sections were cut radial to the tumor, every twentieth section being stained and the rest retained for further investigation. The cornea was intact. The episclera at the external site of the tumor was thickened and infiltrated with fibroblasts and lymphocytes (fig. 4). Normal epithelium covered the defect in part. Embedded in the normal tissue of the anterior third of the sclera, and exclusively under the site of the excised tumor, were large pigmented cells. These corresponded in appearance to the dermal (or choroidal) chromatophores (fig. 5).

Nothing was seen in the sections corresponding to the stellate pigment spots of the cornea except a few clear spots peripherally, where the basal epithelial cells contained a considerable number of vacuoles. It is possible that pigment may have been here; if so, it must have differed from that at the limbus and in the cyst of the iris in being soluble in alcohol. As no tissue was embedded in gelatin for fat staining, we remain uncertain of the nature of the clinically visible corneal pigmentation.

Schlemm's canal beneath the tumor was broad, patent and lined with endothelium (fig. 6). Its posterior wall (on the side of the anterior chamber) consisted of a dense meshwork of six to eight glassy lamellae, whose narrow interstices were filled with chocolate brown pigment. This was of coarser grain than is the pigment usually seen in cases of glaucoma or iritis, and its extension covered the peripheral end of Descemet's membrane (figs. 7 and 8). Nine patent (limbal) vessels could be seen in cross section between Schlemm's canal and the

Fig. 3.—Slit lamp picture of the cyst and satellites as viewed from behind. The brownish cyst is partly covered by the ciliary processes; × 10.

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region of the limbus. Five of these were of considerable size. In longitudinal section (fig. 7) an empty vessel, probably an aqueous vein of Ascher, could be seen to join the canal.

The deeper layers of the pupillary three fourths of the iris were normal. The pigmented layers of the fourth portion on the ciliary side were split to form a rhomboid cyst, measuring 700 by 400 microns (figs. 4 and 6). The cyst was optically empty, and its base was formed by pigmented dilator cells, whose structure was clearly defined. On the pupillary side, the wall of the cyst was thickened and infolded (fig. 6). Several loculi were visible in the wall of the cyst on the ciliary side where it was closely attached to the ciliary processes (fig. 4). Depigmented sections (fig. 9) show the cyst to consist of one row of cubical epithelium, with round nuclei and evenly stained cytoplasm and with no evidence of mucinous changes.

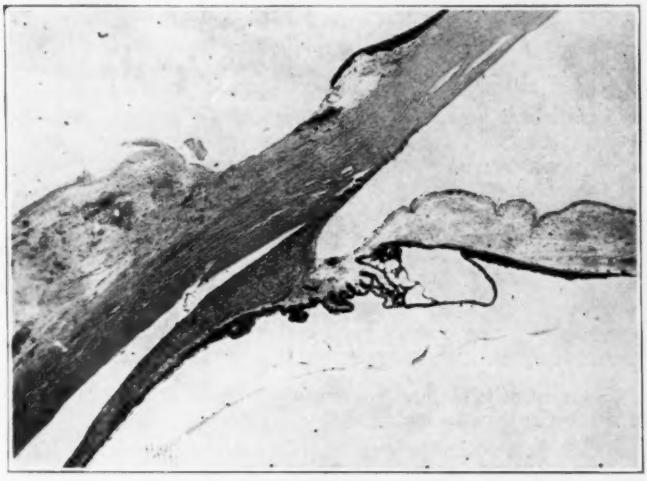

Fig. 4.—Area of melanoma and spontaneous cyst of pigmentary epithelium of the iris, with several chambers (low power). Note the swollen area of the excised episcleral melanoma.

Several other small cysts (fig. 6), lined with one layer of unpigmented epithelium, were observed in the ciliary processes in roughly the same area.

COMMENT

There are two conditions in this eye to be considered: the malignant melanoma of the limbus and the cyst on the posterior surface of the iris. In the absence of operation, disease or injury as a cause in postnatal life, it is probable that the cyst is of congenital origin. As the two lesions are restricted to the same sector of the globe, the chief question concerns their possible causal relationship. Was one lesion in fact caused by the other, or have they a common cause? Previous embryologic and pathologic observations throw some light on this point.

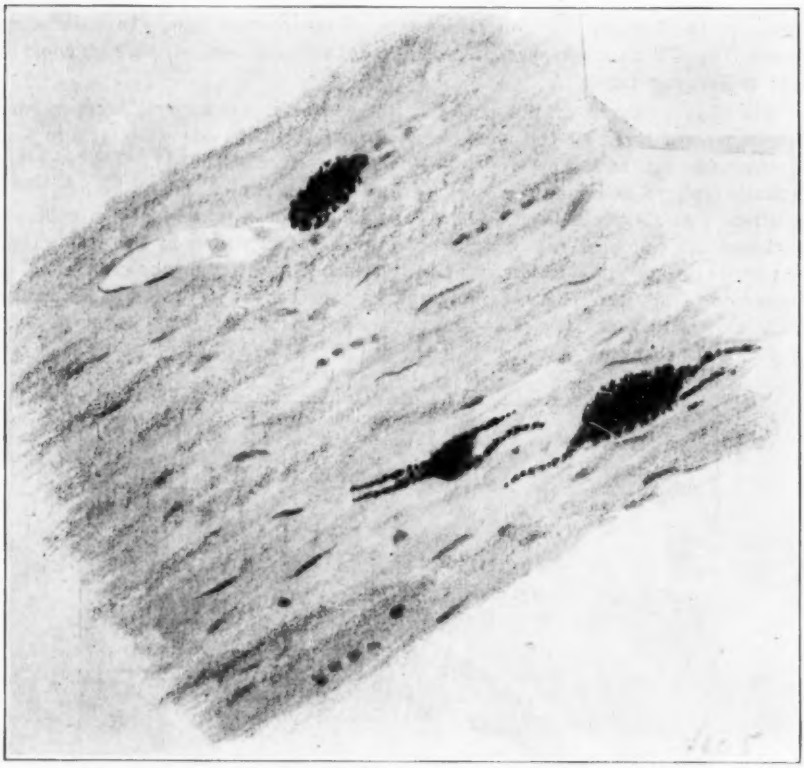

Fig. 5.—Pigment cells of the stroma in the superficial third of the sclera under the site of the excised melanoma; × 300.

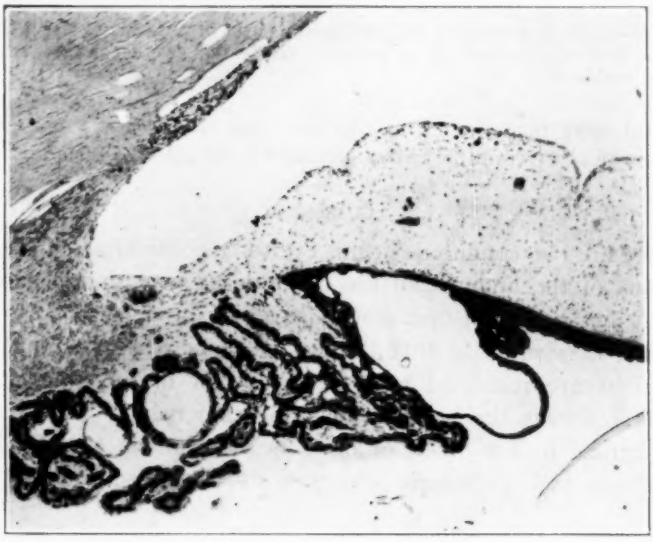

Fig. 6.—Spontaneous cyst of the iris. Note the heavily pigmented meshwork behind Schlemm's canal and the cyst in the ciliary body. Hematoxylin and eosin; × approximately 150.

Spontaneous cysts of the pigmented epithelium are alleged to be rare. Ginsberg [1] recorded 7 cases only in the world literature up to 1928—a figure which Gilbert repeated in 1930 [2] and Duke-Elder [3] increased to 13 in 1940. This does not include the relatively common cysts of ciliary epithelium, which may arise, as indicated by Coats [4] in 1908, from inflammatory adhesions of the epithelium of contiguous ciliary processes or of the ciliary processes and the iris.

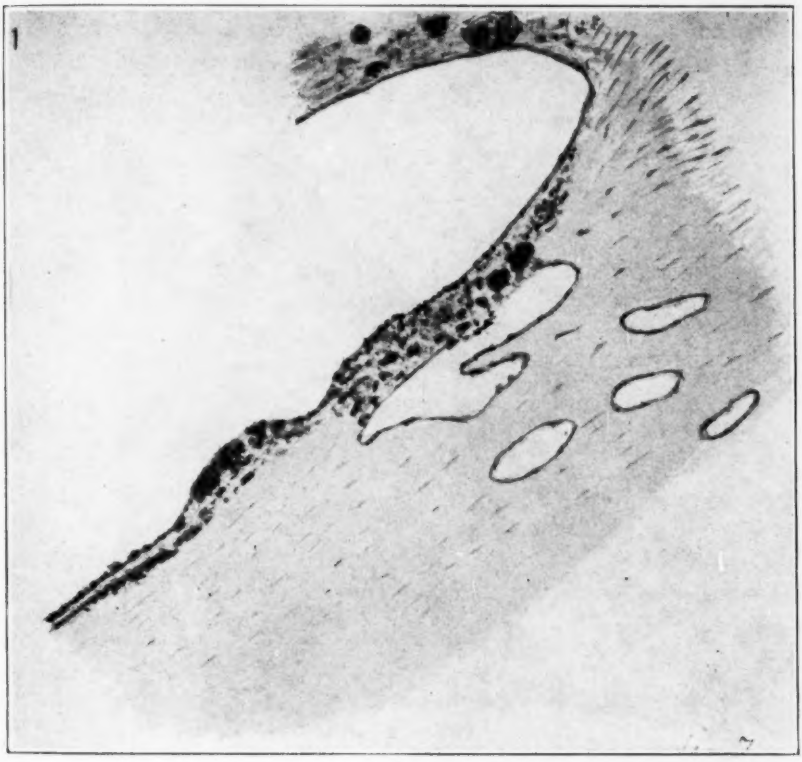

Fig. 7.—Densely packed coarse pigment behind Schlemm's canal, continued toward and enveloping Descemet's membrane. The empty spaces may correspond to aqueous veins of Ascher. The spur hints at their confluence with Schlemm's canal. Hematoxylin and eosin; × 60.

1. Ginsberg, S., in Wessely, K.: Auge, Berlin, Julius Springer, 1928, vol. 1, p. 541.

2. Gilbert, W., in Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, p. 96.

3. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1940, p. 2443.

4. Coats, G.: Roy. London Ophth. Hosp. Rep. **17**:143, 1908.

Our own observation has confirmed this occurrence on several occasions. The much less frequent spontaneous cysts of the posterior surface of the iris may arise by incomplete obliteration of the gap between the two germinal layers of the secondary optic vesicle. This gap exists, under the name of "von Szily's (ring) marginal sinus," normally as late as the fifth month of fetal life.

Gilbert [5] (1910) described a solid, noninfiltrating tumor of the iris of epithelial origin, with multilocular cyst formation in its supramarginal

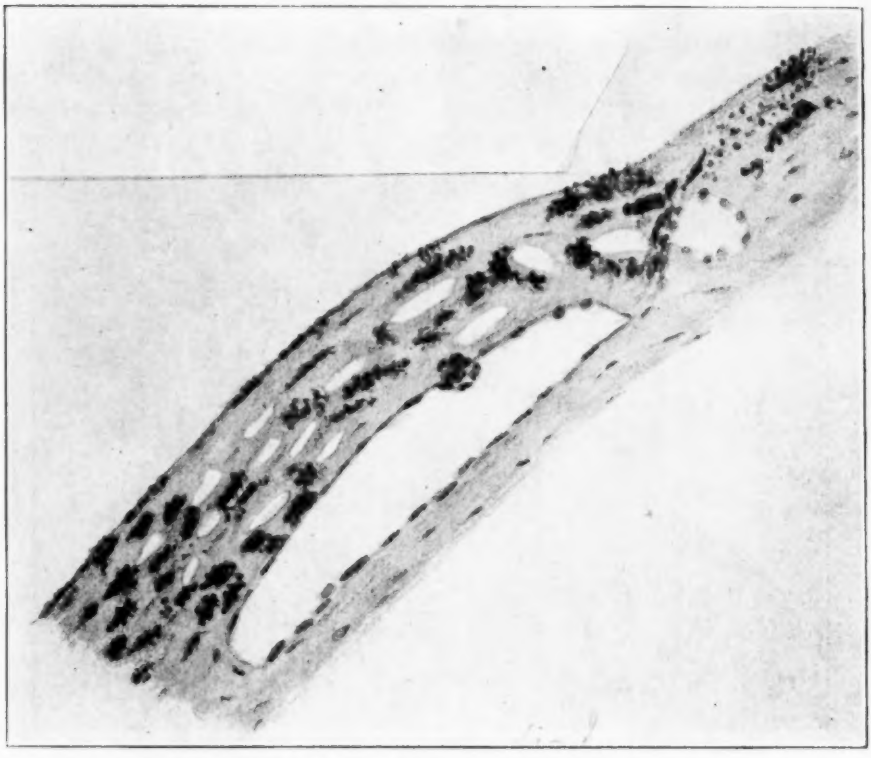

Fig. 8.—Coarse stroma pigment in front of Schlemm's canal, on the tumor side. There is much less on the opposite side of the section. Hematoxylin and eosin; × 300.

portion. He postulated a primary cyst of the iris arising between two layers of the iris, with the tumor as a secondary, nonmalignant development. It is interesting to note that Treacher Collins [6] recorded the occurrence of a small spontaneous cyst between the two pigmented layers of the iris in an eye excised for sarcoma of the ciliary body.

5. Gilbert, W.: Klin. Monatsbl. f. Augenh. **48**:149, 1910.

6. Collins, E. T.: Roy. London Ophth. Hosp. Rep. **13**:41, 1890-1891.

Unfortunately, the short description of this case does not include reproductions of the slides.

The discovery in our case of a uveal cyst with its satellite patches of brown pigment on the posterior surface of the iris underlying the episcleral melanoma was quite unexpected. This particular area was being examined for evidence of possible melanotic infiltration.

As the slit lamp excluded similar changes elsewhere, one is presented with the coexistence of two relatively rare pathologic conditions in one and the same sector of the eye. Is this a matter of chance only, or is it possible to find a common cause for these two new growths of fundamentally different type? Our ultimate conclusion is that the

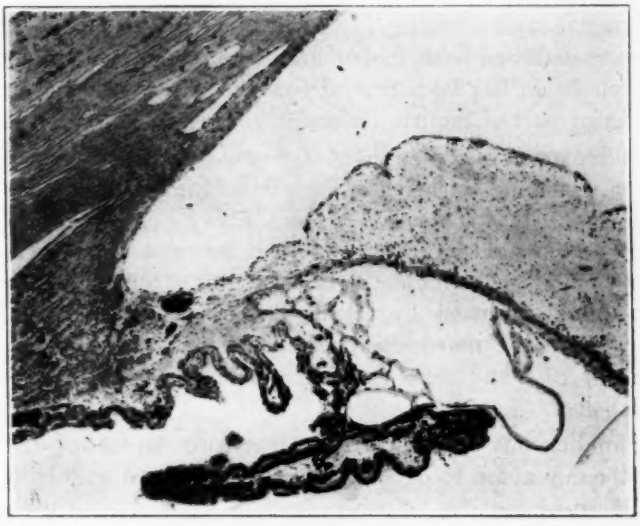

Fig. 9.—Depigmented epithelium (low power).

internal and external abnormalities we have described were all due to antenatal disturbance, probably inflammatory.

Of course, one cannot be certain at what developmental stage this arose, but it probably antedated formation of the anterior chamber and the complete closure of the cleft between the two germinal layers (16 mm.). The changes we have postulated might well occur then. We assume that this process was intrauterine uveitis.

It is common knowledge that the fetal uvea may be affected by septic processes (in the widest sense) in the mother due to a variety of bacteria and viruses. During the past twenty years inexplicable genetic aberrations of a chromosomal character have borne almost the entire blame for congenital abnormalities, and the part played by

fetal inflammation has been greatly underestimated. Jankû's [7] observations (1923) regarding that perennial puzzle, coloboma of the macula, met with little support until American authors confirmed his claims by demonstrating that such intrauterine changes could be caused by systemic toxoplasmosis of the mother. The embryonic damage due to maternal rubella in the first two months of pregnancy has attracted the attention of the medical world since the publication of Gregg's [8] paper in 1941. This article is of particular interest to the ophthalmologist in view of the high incidence of congenital cataract associated with these deformities (Long and Danielson [9]).

One might expect that such an inflammatory process could produce the following changes:

1. Extension and incomplete obliteration of the space between the two germinal layers leading to cyst formation, the posterior (retinal) layer being detached from the anterior (dilator) layer (as in our case). This explanation has been accepted as the basis of congenital cysts of the posterior part of the iris (Mann [10]).

2. Inflammatory dilatation of the vascular element in the tunica vasculosa lentis and intrusion of such vessels into the capsular sac, with disorganization of the developing lens fibers. Loewenstein [11] (1920) explained the origin of congenital cataract and its association with persistent pupillary membrane in this way, his theory being subsequently confirmed and accepted by Riedel [12] (1922), Pesme [13] (1927) and others. It will be noted that iceberg-like cataracts occurred in both eyes in our case and were associated with posterior synechias in one (the left) eye.

3. Simultaneous disturbances in the two layers of the anterior part of the cup about to develop into the pigmented epithelium and the dilator layer of the iris. Such potentially pigmented neuroectodermal cells, when displaced as a result of fetal iritis, might give rise to a postnatal melanoma of the limbus.

Although the explanation offered of the association observed in our case may be an oversimplification, we do not feel that it is too far fetched. It is our hope that application of the technic of examination employed in this case may lead to the discovery of similar cases, which routine histologic technic may easily overlook.

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7. Janků: Časop. lék. česk. **67**:1021, 1923.
8. Gregg, N. M.: Tr. Ophth. Soc. Australia **3**:35, 1941.
9. Long, J. C., and Danielson, R. W.: Cataract and Other Congenital Defects in Infants Following Rubella in the Mother, Arch. Ophth. **34**:24 (July) 1945.
10. Mann, I.: Developmental Abnormalities of the Eye, London, Cambridge University Press, 1937.
11. Loewenstein, A.: Arch. f. Ophth. **103**:37, 1920.
12. Riedel: Klin. Monatsbl. f. Augenh. **69**:482, 1922.
13. Pesme, P.: Arch. d'opht. **44**:620, 1927.

SUMMARY

A malignant episcleral melanoma, a spontaneous cyst of the posterior tissue of the iris and a heavy deposit of pigment in the meshwork behind Schlemm's canal were observed in the same sector of the eye of a woman aged 33, in addition to a bilateral congenital cataract.

It is suggested that fetal inflammation simultaneously produced the cyst, by preventing complete closure of the cleft between the two layers of the optic vesicle, and the melanoma, by displacement of the pigment-storing cells of the outer germinal layer. The congenital cataract and the heavy pigmentation of the meshwork behind Schlemm's canal probably originated in the same inflammatory process.

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ATROPHY OF THE OPTIC NERVE FOLLOWING HEMORRHAGE

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AMBLYOPIA following acute loss of blood is an old, but relatively rare, disease. An average of 1 case a year has been reported in the world's literature in the past twenty-five years. Colonel Derrick Vail saw only 4 cases of such visual loss in his vast experience in military ophthalmology in Europe in World War II.

That the pathogenesis of atrophy of the optic nerve following hemorrhage is imperfectly understood is evidenced by the numerous and varied theories (recently summarized by Cox [1]) that have been put forth to explain the condition. Knowledge of its pathogenesis is limited because few such blind eyes have ever been examined microscopically. The few histologic studies made have revealed edema of the retinas and optic disks with degenerative changes in the retinal ganglion cells, similar to those associated with quinine poisoning. In addition, Goerlitz [2] observed foci of degeneration in the optic nerve behind the lamina cribrosa. If conclusions are to be drawn from these microscopic observations, the seat of the disease must lie in the ganglion cells or in their fibers behind the lamina cribrosa, for edema of the optic disk and retina seems insufficient to cause optic nerve atrophy within a few days or weeks. This view is supported by the animal experiments of Holden [3] and of Uhthoff [4] and leads to the most tenable explanation of the disease, namely, that retinal ischemia causes degeneration of the ganglion cells, the third retinal neurons, the axons of which constitute the fibers of the optic nerve.

Objections to this theory are the following facts: 1. Hemorrhages of all kinds are frequent, whereas associated visual loss is rare. 2. There is

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1. Cox, R. A.: Amblyopia Resulting from Hemorrhage, Arch. Ophth. **32**: 368-371 (Nov.) 1944.

2. Goerlitz, M.: Blindness Following Severe Loss of Blood: Histologic Study of a Case, Klin. Monatsbl. f. Augenh. **64**:763-782, 1920.

3. Holden, W. A.: The Pathology of the Amblyopia Following Profuse Hemorrhage, Arch. Ophth. **28**:125-134, 1899.

4. Uhthoff, W.: Blindness and Changes in the Fundus Associated with Anemia, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **43**:204-212, 1922.

a time lag between the cessation of bleeding and the appearance of blindness, with visual trouble developing between three and sixteen days after the hemorrhage in 39.2 per cent of the cases (Singer [5]). 3. In 15 per cent of the cases the blindness is unilateral. 4. The fundus picture is variable, showing marked papilledema in some cases and no visible retinal changes in other cases. 5. The degree of visual loss does not appear to be dependent on the amount of blood lost, for in some cases this is reported to be small. Furthermore, repeated small hemorrhages are more likely to cause this unfortunate complication than a single large hemorrhage. In view of these objections, it seems plausible that some factor other than ischemia may be playing a role in producing the atrophy of the optic nerve. The process is independent of age, race or sex; and since it rarely, if ever, occurs in a healthy person, Zentmayer [6] stated the opinion that in all such cases some underlying factor had previously reduced the vitality of the retinal ganglion cells.

Since routine and accurate blood counts and determinations of blood pressure were not practiced before the turn of this century, the 190 cases reported up to 1901 which were exhaustively reviewed by Singer [5] do not permit an evaluation of the importance of these factors in the causation of the amblyopia following hemorrhage. The 24 cases of Zentmayer [6] and the 12 cases of Harbridge [7] represent the literature from 1901 to 1924, but even in this group few blood counts or blood pressure readings were made; and, indeed, many of the patients were not seen by the ophthalmologist until weeks or months after the onset of blindness. The case reports since then are more complete in this respect but leave much to be desired. Inasmuch as the histologic studies of this disease are so meager, clinical and laboratory data must be relied on more heavily in any attempt to shed light on the mechanism of atrophy of the optic nerve. We were able to study a case of partial blindness following severe hemorrhage within twenty-four hours after the patient's complaint of visual loss, and the clinical and laboratory findings are described in some detail.

REPORT OF A CASE

On Dec. 1, 1944, E. W. K., a 34 year old white man, a van driver, was admitted to the Mount Alto Hospital, Washington, D. C., by ambulance for the treatment of a bleeding peptic ulcer. He had been under the care of his private physician for four months, when, on Nov. 27, 1944, he had vomited a large amount of blood and on the following day had fainted. The past history revealed that on induction into the Army, on Oct. 24, 1942, vision was 20/20 in each eye. During his military ser-

5. Singer, K.: Blindness Following Loss of Blood, Beitr. z. Augenh. **53**:1-103, 1904.

6. Zentmayer, W.: Visual Disturbances from Distant Hemorrhages, J. A. M. A. **59**:1050-1055 (Sept. 21) 1912.

7. Harbridge, D. F.: Optic Atrophy Manifested by Visual Disturbance Following Distant Hemorrhage, Am. J. Ophth. **7**:192-196, 1924.

vice, part of which he spent in Africa, he was hospitalized on several occasions because of painful, stiff joints. The hospital records described him at various times as "nervous, apprehensive and slow to answer questions," with a constant coarse tremor of the hands. A history was elicited of alcoholic sprees which lasted a week at a time and had occurred about every two months since the age of 25, although his record was clean in this respect during his military experience. He was discharged from the Army on Aug. 11, 1943, with the diagnosis of nonsuppurative rheumatoid arthritis. Roentgenograms of the joints, the sedimentation rate and the blood count were repeatedly normal, and the Kahn reaction of the blood was negative.

Examination on admission revealed a tall, pale, confused, dyspneic, restless young man, with a precordial systolic murmur. The systolic blood pressure was 130, and the diastolic pressure was unobtainable. The temperature was 98.4 F.[8a]; the pulse rate, 104, and the respiratory rate, 26. Treatment consisted of sedation, administration of antacids and atropine and giving fluids by mouth. The next day the blood count revealed 1,220,000 red cells with a hemoglobin reading of 25 per cent and 11,000 white cells. The urine was normal, and the Wassermann and Kahn reactions of the blood were negative. A transfusion of 500 cc. of citrated blood was given. On the following day the temperature had risen to 103.8 F. On December 4 the patient complained of terrific frontal headache and sudden blindness in the "left side of his left eye." The temperature was 100.8 F.; the pulse rate was 124, and the respiratory rate, 26, per minute. The blood pressure was 110 systolic and 0 diastolic. The red blood cell count was 1,550,000; the hemoglobin concentration, 29 per cent, and the white cell count, 6,800.

Ophthalmologic consultation next morning revealed that the pupils were round and equal, reacting well to light and in accommodation. The extraocular movements were good. Determination of the visual fields with the confrontation test revealed temporal hemianopsia in the left eye. The right optic disk was pink, with blurred but not elevated margins. The left disk was enlarged, with blurred edges and a slight elevation of less than 1 D. The nasal half of the disk was definitely pale. In each fundus the arteries were extremely narrowed and the veins widely dilated, with conspicuous arteriovenous nicking. Numerous superficial hemorrhages and some white spots were scattered about bilaterally. The maculae were normal.

The patient continued to complain of severe frontal headache and that night began to vomit "coffee grounds" material and fresh blood clots, falling to the floor in his weakness when he attempted to get out of bed. On December 6 he became disoriented and incontinent of urine. The temperature was 103.2 F., the pulse rate was 140, and the respiratory rate 32, per minute. The blood pressure was 108 systolic and 0 diastolic. The red blood cell count was 900,000; the hemoglobin concentration, 15 per cent, and the white cell count, 11,650. He was given 500 cc. of blood in the morning and 500 cc. of blood in the afternoon. The next morning he complained of almost complete blindness in the left eye. Examination now revealed marked pallor of the entire left disk, with visual acuity in that eye reduced to the perception of moving shadows at 6 inches (15 cm.). Otherwise the fundi were unchanged. Another transfusion of 500 cc. of blood was given. On December 8 he was still acutely ill. The temperature was 101 F.; the pulse rate was 132, and the respiratory rate 28, per minute. The red cell count was 970,000; the hemoglobin concentration, 15 per cent, and the white cell count, 21,600. Another 500 cc. of blood was given. Two days later he complained of some

8a. The temperatures listed are the highest for the given day.

decrease in the vision of his right eye, but the appearance of the fundus was unchanged. Blood pressure was 120 systolic and 60 diastolic. At night he climbed over the side rails of his bed and tried to leave the hospital. By December 21, all bleeding from the ulcer had ceased, and he had become afebrile. Fluoroscopic examination revealed a duodenal ulcer, and this finding was confirmed by roentgenograms.

Examination on the day of discharge, on Feb. 3, 1945, revealed that the pupils were round and equal. They reacted well in accommodation. The right pupil reacted well to light; the left pupil reacted slowly to light through a narrow arc and then dilated rapidly.

Right Eye: The optic disk had a hazy superior margin. Its upper half was very pale. The arteries were slightly narrowed, with an artery to vein ratio of 1 : 2 and with moderate arteriovenous nicking. A patch of white substance lay above the superior temporal vein. The macula was normal. Determination of the visual field revealed a contraction of the temporal half to 50 degrees and a defect in the inferior nasal quadrant. Visual acuity was 20/30 + 2, correctible to 20/15—1.

Left Eye: The disk was very pale, with hazy and irregular but not elevated edges. Some of the arteries were narrow, with an artery to vein ratio of 1 : 3, but the pronounced arteriovenous nicking and dilatation of the veins had disappeared and no white spots or hemorrhages were present. The visual field could not be determined because visual acuity was reduced to light perception but not projection.

The patient was quiet but tremulous, with slow movements and coherent but hesitant speech. The blood count revealed 3,740,000 red cells and a hemoglobin content of 78 per cent.

On examination in the outpatient department three months later, the patient stated that his vision had remained "about the same." The ophthalmologic findings also were essentially unchanged. The pupils were round and equal but dilated, measuring 6 mm. Visual acuity of the right eye was 20/20—2 with ability to read Jaeger type 1, and was correctible to 20/15—1. The disk was flat and somewhat pale, with only the inferior quarter normally pink. The remainder of the fundus was unchanged. The defect in the inferior part of the field was no longer present, so that there was now an almost concentric contraction of the field to five-eighths normal size. The visual acuity and appearance of the fundus of the left eye were unchanged, and the disk was totally white and flat, with rough edges and pronounced narrowing of some of the retinal arteries. The peculiar mental status, with the slow and retarded reaction time and hesitant answers to the simplest questions, was still present. Roentgenograms of the joints showed nothing abnormal. The ophthalmologic findings on reexamination of the patient, on Nov. 5, 1945, were identical with those already described.

COMMENT

Analysis of this case reveals sufficient cause for impaired nutrition to the retina. Within a few days after a massive hematemesis the patient had lost four fifths of his red blood cells, causing a reduction in the hemoglobin to 25 per cent, and encroaching seriously on the oxygen-carrying capacity of the blood. Because of continued bleeding this blood picture did not improve for thirteen days, in spite of transfusions totaling 2,500 cc. of blood. Failure of the peripheral circulation was present, as evidenced by the weak, rapid pulse and the fact that the diastolic pressure was unobtainable for a week, even though the systolic

pressure was maintained fairly well. Wolff [8] postulated that this type of amblyopia was due to spasm of the retinal arterioles, resulting from lack of oxygen, while Duggan [9] attributed it to vasoconstriction produced by an increased output of epinephrine stimulated by the hemorrhage. Whichever one of these mechanisms may have been operating to produce the narrowing of the arteries seen in this patient's fundi, the effect of the decrease in the caliber of the arteries was to increase further the ischemia of the visual elements of the retina. In addition, two days before the patient's visual loss a fever developed, which lasted three weeks. Regardless of its cause (and no focus of infection was ever found), fever causes an increased metabolic rate in all tissues, and thus the need of the retinal cells for oxygen increased at a time when their supply of oxygen was already deficient. In this connection it is interesting to note that Langdon's [10] patient had a rise in temperature to 103 F. on the day preceding her visual loss and Long's [11] patient had a fever of unknown origin three days before the onset of optic nerve atrophy. The delirium exhibited by the patient for two weeks not uncommonly occurs after severe, acute loss of blood and is generally attributed to cerebral anoxemia. As a result of this mental confusion, the anemia of the brain and the retinal cells was increased, for the patient would not lie flat in bed.

The presence of pallor of part of the optic disk of the right eye a few weeks after the hemorrhage may be attributed to degeneration of the retinal ganglion cells with subsequent gradual ascending atrophy of the optic nerve fibers. The almost concentric contraction of the peripheral field supports this view, for the periphery of the retina has the poorest blood supply. But how is one to explain the atrophy of the medial optic nerve fibers in the left eye occurring suddenly a week after the onset of bleeding and followed two days later by atrophy of the temporal optic nerve fibers? These findings point strongly to direct involvement of the optic nerve fibers in the pathologic process. According to Duke-Elder,[12] the optic nerve fibers at the disk receive their blood supply from recurrent branches of the central retinal artery, supplemented by anastomoses with a few of the short posterior ciliary

8. Wolff, E.: The Causation of Amblyopia Following Gastric and Other Hæmorrhages, Tr. Ophth. Soc. U. Kingdom **55**:342-349, 1935.

9. Duggan, W. F.: Clinical Vascular Physiology of the Eye, Am. J. Ophth. **26**:354-368, 1943.

10. Langdon, H. M.: Amaurosis After Uterine Hemorrhage with Restoration of Vision Following Transfusion, Arch. Ophth. **10**:99-102 (July) 1933.

11. Long, A. E.: Amaurosis Following Nasal Hemorrhage, Am. J. Ophth. **26**: 1179-1182, 1943.

12. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1944, pp. 106 and 136.

arteries (of the circle of Zinn). These nutrient arteries of the optic nerve are small end arteries, like those of the retina. Presumably, if severe hemorrhage can cause a decrease in the quality and quantity of the blood supply to the retina, it can have a similar effect on the blood supply to the optic nerve, with resulting ischemic degeneration of the fibers of the nerve head. The observation of Goerlitz [2] of foci of degeneration behind the lamina cribrosa would tend to uphold this view.

With this evidence in mind, it would appear that the burden of proof should rest on him who states that ischemia is not the cause of the atrophy of the optic nerve following hemorrhage. Moreover, the objections to the theory of retinal ischemia mentioned in the introduction are not applicable in this case. While acute hemorrhages of all kinds are frequent, few are so severe that they produce a drop in the hemoglobin to 15 per cent. According to the medical literature, only rarely does a human being survive such extreme exsanguination and so acute an anemia — a fact which may account for the rarity of posthemorrhagic atrophy of the optic nerve. Also, the disease was bilateral and occurred during, and not after, a prolonged bout of bleeding. Nevertheless, like Zentmayer, one cannot but feel that a predisposing factor is present in this malady. Terson [13] emphasized the frequency with which cerebral phenomena accompany posthemorrhagic blindness. These he described as coma, violent head pains, polyneuritis, aphasia, amnesia, oculomotor paralyses and deafness. Since the metabolism of the brain is identical with that of the inner, or cerebral, part of the retina, it is tempting to attribute the pathologic changes in the two organs to the same factor, namely, ischemia. This would furnish a ready explanation for cases like that of Langdon,[10] in which there developed "definite cortical involvement," coincident with atrophy of the optic nerve. While my patient, after the clearing of his mental cloudiness, showed none of the neurologic manifestations mentioned by Terson, he did show persistent signs of neuropsychiatric disease, which, as the history revealed, long antedated the duodenal hemorrhage. His peculiar mental status presupposes a lack of integrity in the central nervous system, and this element cannot be excluded as a predisposing factor in the causation of the optic nerve atrophy. Other examples of the occurrence of this malady in patients with a history of preexistent psychiatric disease are the reports of Harbridge,[7] who described his patient as "irritable and erratic," and Robertson,[14] whose patient had

13. Terson, A.: Pathogenesis and Treatment of Visual Disturbances After Loss of Blood, Ann. d'ocul. **159**:23-65, 1922.

14. Robertson, C. K.: Sudden Blindness Following Gastro-Intestinal Hemorrhage, Edinburgh M. J. **48**:414-418, 1941.

been retired from all work five years previously because of "a marked mental disturbance."

CONCLUSIONS

During a massive hemorrhage from a duodenal ulcer a 34 year old white man experienced partial atrophy of the optic nerve in one eye, with later development in the other eye. Repeated determinations of the blood count and the blood pressure during this illness revealed a persistent state of peripheral circulatory failure and such an extreme acute anemia that ischemia is considered the chief etiologic factor in the production of the pathologic changes observed. The ophthalmologic findings suggest that ischemia can cause atrophy of the optic nerve either by producing degeneration of the retinal ganglion cells or by direct destruction of the optic nerve fibers in the region of the lamina cribrosa. The former mechanism best explains the slow, delayed onset of partial atrophy of the right optic nerve of this patient, whereas the latter mechanism best explains the rapid, early onset of almost total optic atrophy of the left optic nerve.

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TREATMENT OF LEWISITE BURNS OF THE EYE WITH DIMERCAPROL ("BAL")

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EXPOSURE of the eye to relatively small quantities of liquid or vapor lewisite (betachlorovinyldichloroarsine) produces a devastating ocular lesion. As will be demonstrated later, the progressive nature of such a burn is caused by the arsenical component of this war gas. To decontaminate the tissues of arsenic after exposure to lewisite, English workers synthesized 2,3-dimercaptopropanol (CH_2-SH:CHSH:CH_2OH), now called dimercaprol ("BAL"). The work pursued in the laboratories of the Wilmer Ophthalmological Institute was devoted to the determination of the optimum conditions for the use of this antidote, its mode of action and its limitations. These experiments will be discussed under the following headings: (1) the distinctive clinical and pathologic characteristics of lewisite burns of the eye with respect to mode of action, rate of penetration and the time at which irreversible histologic changes first develop; (2) the rate of penetration and persistence of arsenic in the tissues after lewisite burns in relation to the decontaminating action of dimercaprol, and (3) the toxicity and therapeutic efficiency of dimercaprol.

PATHOLOGIC CHARACTERISTICS OF LEWISITE BURNS OF THE EYE

During the course of this work, more than 600 rabbit eyes were exposed to lewisite. The characteristics outlined here were obtained from clinical observations and histologic sections of this material, including burns of all degrees of severity produced by either liquid or vapor lewisite. In general, lewisite burns were characterized by rapid tissue necrosis, pronounced conjunctival and corneal edema and intense exudation. Histologic evidence of corneal damage was found ten minutes after a thirty second exposure to saturated lewisite vapor at 23 C. (fig. 1 *A*). At this time, the basal cells of the corneal epithelium were peglike, somewhat edematous and partially detached from the underlying stroma. The cells of the corneal stroma showed small horizontal clear spaces beneath some of the nuclei, and after thirty minutes the nuclei were

This work was done under a contract, recommended by the Committee on Medical Research, between the Office of Research and Development and the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

partially broken up (fig. 1*B*). At the end of ten minutes the corneal endothelium was partially disintegrated and serum and fibrin were present in the anterior chamber.

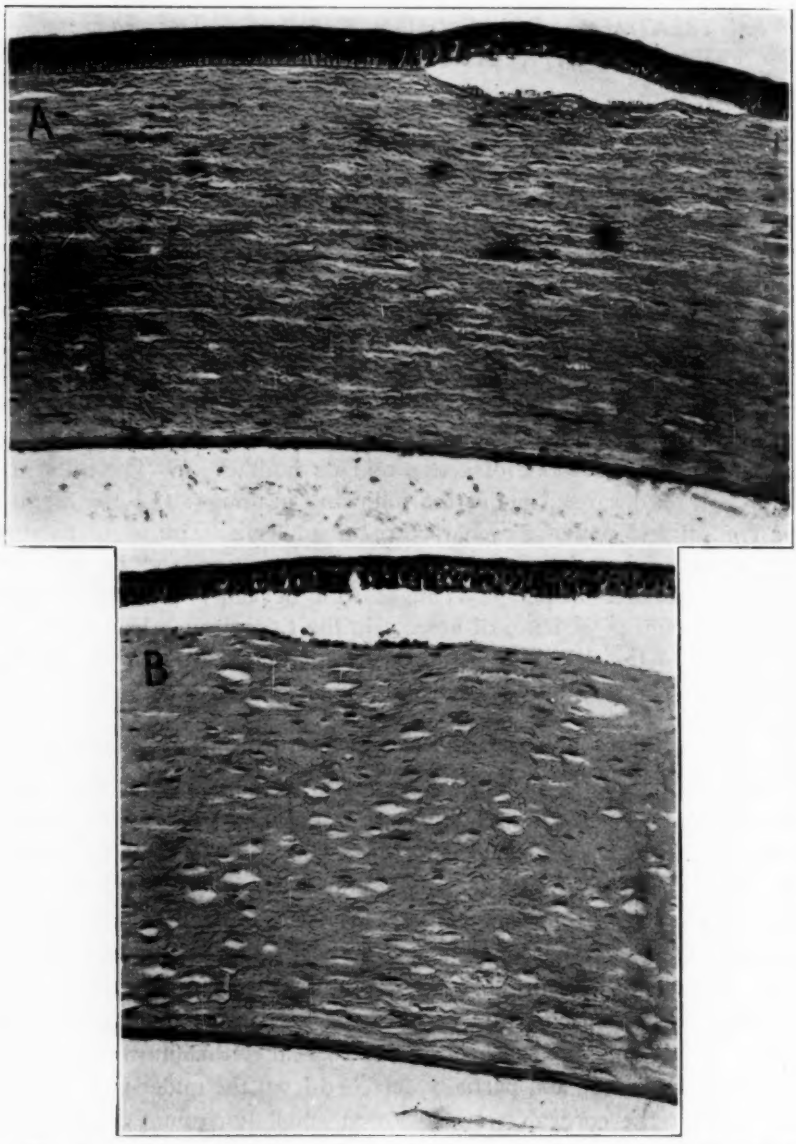

Fig. 1.—Sections of rabbit cornea (*A*) ten minutes, and (*B*) thirty minutes, after exposure for thirty seconds to saturated lewisite vapor at 22 C.

The application of 0.1 mg. of liquid lewisite to the surface of the cornea produced instantaneously a dense, circumscribed opacity at the

point of contact. Within five to ten minutes, the localized opacification extended down through one half of the corneal stroma, and small vesicles began to appear on the surface of the involved area. The walls of limbal blood vessels near the site of application were destroyed, producing petechial hemorrhages, thromboses and ischemia (fig. 2). Within a few days after the application of liquid lewisite to the limbus, a sector-shaped area of hemorrhage and necrosis of the iris could usually be distinguished directly beneath the site of application. This evidence of deep and localized penetrability of the agent could also be demonstrated histologically.

Except for the localized areas of damage produced by the liquid, the subsequent courses of burns produced by vapor and those by liquid lewisite were remarkably similar. Briefly, the changes consisted of

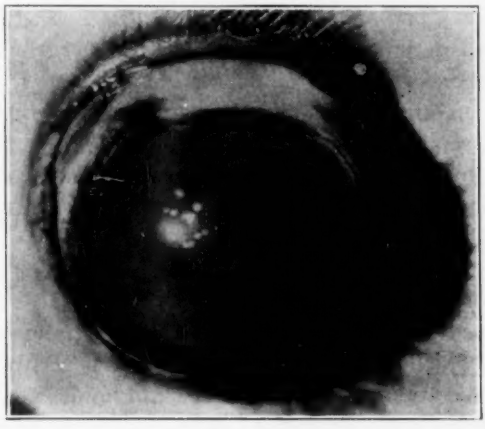

Fig. 2.—Rabbit eye eleven days after the instillation of 0.1 mg. of liquid lewisite on the limbus at 12 o'clock. The small round site of application is still visible, and the surrounding area is ischemic. Corneal vascularization does not proceed from this area.

marked conjunctival edema with petechial hemorrhages and thrombosis of the blood vessels; complete desquamation of the corneal epithelium and edema, purulent infiltration, vascularization and ulceration of the cornea; fibrinous iritis; occasionally secondary glaucoma with staphyloma of the cornea, and cataract.

The Acid Burn Component of Lewisite Burns.—The dense superficial area of opacification which appears instantaneously on contact of liquid lewisite with the moist surface of the cornea is probably produced by hydrochloric acid which is liberated by hydrolysis of the lewisite.

$$ClCH:CH.AsCl_2 + H_2O = ClCH:CH.AsO + 2HCl$$

Evidence for this view was obtained by the injection of indicator dyes

into beef corneas beneath the sites where lewisite was subsequently applied. It was found that all dyes immediately changed to a color indicative of an acid reaction within a radius of 2 to 4 mm. of the site of the lewisite droplet. Thymol blue became red in this area, indicating a local p_H of less than 1.3. Hydrochloric acid solutions of less than p_H 2.5 have been found previously to produce corneal damage.[1]

It will be shown later that the use of dimercaprol effectively prevents the progressive development of corneal lesions produced by lewisite. However, the opaque site of application is not altered by such treatment, affording additional evidence that lewisite produces an acid burn, similar to other acid burns, which cause rapid and irreversible precipitation of corneal proteins.[1] Similarly, the superficial corneal haze immediately visible following exposure to lewisite vapor is probably due to the acid portion of hydrolyzed lewisite.

The Arsenical Component of Lewisite Burns.—All the later characteristics of a lewisite burn of the eye can be produced by the instillation of a dilute solution of lewisite oxide (ClCH:CH.AsO). It was found that a number of other compounds containing trivalent arsenic (e. g., sodium meta-arsenite, phenylarsine oxide and oxophenarsine hydrochloride) were more toxic on intracorneal injection of dilute solutions than compounds containing pentavalent arsenic (e. g., sodium arsenate and tryparsamide).

In summary, lewisite hydrolyzes immediately on contact with the moist surface of the eye, producing a superficial acid burn followed by a progressive lesion of the cornea because of the toxicity of the lewisite oxide, which contains trivalent arsenic. Pathologic changes in all tissues of the anterior ocular segment can be detected histologically within ten minutes after exposure, indicating deep penetrability and rapid necrotizing action of this toxic arsenical.

RATE OF PENETRATION AND PERSISTENCE OF ARSENIC IN THE EYES OF RABBITS BURNED WITH LEWISITE

The rate of penetration and the persistence of toxic arsenical material in the cornea and aqueous following exposure to lewisite were determined in order to establish time limits during which either a 100 per cent efficient surface-decontaminating agent or an ideal penetrating agent would be effective.

Materials and Methods.—Rabbit corneas were exposed to lewisite, either by the instillation of 0.1 mg. of the liquid onto the center of the cornea or by exposure for thirty seconds at 22 C. to saturated vapor. The lids were closed within thirty seconds after exposure. Then, at varying intervals, the corneas were excised, washed briefly with saline solution and dropped into a solution of arsenic-free

1. Friedenwald, J. S.; Hughes, W. F., Jr., and Herrmann, H.: Acid Burns of the Eye, Arch. Ophth. **35**:98 (Feb.) 1946.

nitric acid preparatory to analysis for arsenic by the method of Chaney and Magnuson.[2] With this method, "blank" control tissues may show as much as 0.5 microgram of arsenic. The experimental error for determination of such small amounts of arsenic is about 10 per cent, but any value over 1.0 microgram indicates the definite presence of arsenic. Microchemical estimations of arsenic in the aqueous were also made after the application of 0.1 mg. of lewisite to the rabbit cornea near the limbus. Approximately 0.2 cc. of aqueous could be obtained by aspiration with a tuberculin syringe and a 26 gage needle. For specimens taken at ten minutes or before, the lids were not closed after the application of

TABLE 1.—*Scale for Grading the Severity of Ocular Lesions Produced with Lewisite*

Cornea	Maximum Grade
Density of corneal opacity	8
+ 2 = definite opacity	
+ 4 = blurs iris pattern	
+ 6 = blurs outline of pupil	
+ 8 = completely opaque	
Area of corneal opacity	4
Corneal vascularization (intensity and length)	4
Ulceration	4
+ 1 = staining with fluorescein	
+ 2 = ulceration visible without magnification	
+ 3 = deep slough or marked thinning of cornea	
+ 4 = perforation = 100% corneal lesion regardless of other values	
Edema	4
Total points for estimation of corneal lesion = 24 = 100%	
Duration of corneal opacity	4
+ 1 = 1-3 days	
+ 2 = 4-6 days	
+ 3 = 7-13 days	
+ 4 = 14 days and over	
Conjunctiva	
Redness	2
Edema or chemosis	3
Necrosis (petechial hemorrhages or ischemia)	2
Mucopurulent discharge	2
Iritis	3
+ 1 = small pupil, photophobia or positive aqueous ray	
+ 2 = congestion or thickening of iris	
+ 3 = exudation	
Total Points for Estimation of Ocular Lesion = 40 = 100%	

"Maximum ocular reaction" indicates the sum of the maximum values for each symptom during the course of observation and conversion of the total to a percentage figure. This gives an index of the acuteness of the reaction.

"Final corneal opacity" indicates the sum of the values of the corneal symptoms on the last day of observation, a total of 24 points, or corneal perforation representing a 100 per cent lesion. If the cornea became clear, the day on which this occurred is recorded.

lewisite, and the aqueous was withdrawn through a point in the cornea most distant from the site of application. A control analysis indicated that the needle did not become contaminated by any arsenic which might conceivably have spread over the conjunctival or corneal surface.

A second series of experiments was performed to determine the persistence of toxic material on the surface of the cornea, in the cornea or in the aqueous. Direct contact transfers were made from the surface of the exposed eye to the surface of a normal rabbit eye. In order to determine whether the arsenic found within the cornea was still toxic, pressed juice of lewisite-burned corneas was

2. Chaney and Magnuson, H. J.: Indust. & Engin. Chem. (Anal. Ed.) **12:** 691, 1940.

injected intracorneally into the normal rabbit eyes. The juice of 2 to 4 rabbit corneas previously burned with 0.1 mg. of lewisite was expressed by means of a hydraulic press developing 15,000 pounds of pressure per square inch (1,060 Kg. per square centimeter). Residual cornea tissue consisted only of a dry membrane of tissue paper thinness. The juice was then diluted with 1 cc. of saline solution per cornea and passed through a Seitz bacterial filter. All cultures taken of these filtrates were sterile. One-tenth cubic centimeter of this solution was then

TABLE 2.—*Microchemical Analysis of Arsenic in Cornea Following Lewisite Burns of the Rabbit Eye*

Dose of Lewisite	Time After Application	Amount of Arsenic, Micrograms
Controls (no lewisite).....	.....	0 — +0.5
0.1 mg. liquid lewisite.....	2 min.	6.5
		4.1
		6.5
		4.2
		3.7
		2.7
		4.7
	30 min.	1.2
		1.6
	1 hr.	0.7
		2.9
	2 hr.	1.3
	4 hr.	1.8
	11 hr.	0.5
	18 hr.	0.0
	24 hr.	1.7
		0.5
		0.5
	2 days	0.0
	4 days	0.9
Saturated vapor, 22 C. for 30 sec.....	2 min.	2.7
		2.9
		3.3
	30 min.	1.2
	1 hr.	1.9
	4 hr.	1.3
	24 hr.	0.8

used for intracorneal injection into normal rabbits. Injection of 0.1 cc. of normal corneal juice does not result in any residual corneal opacification after twenty-four hours. The toxicity of the aqueous at varying intervals after exposure to lewisite was tested by direct intracorneal injection into normal rabbits. The reactions produced by such intracorneal injections were graded according to a numerical evaluation of the symptoms produced, thus giving an index of the toxicity of the material tested (table 1).

Persistence of Toxic Material on the Surface of the Cornea.—After the instillation of 0.1 mg. of liquid lewisite on the surface of fresh surviving beef eyes, residual toxic material could still be transferred directly

to rabbit eyes after two and one-half hours. To approximate conditions which would follow a splash burn in a normal human eye, the lids of rabbit eyes were closed within thirty seconds after the instillation of the liquid lewisite, and direct contact transfers to normal rabbit eyes were made at varying intervals thereafter. Transfer after one minute resulted in a 10 per cent reaction in the recipient eye, with clearing of the cornea in three days. Transfer after two to four minutes produced only minor transient conjunctival symptoms, without corneal damage. No toxic material could be transferred after four minutes had elapsed.

Two minutes after exposure to lewisite vapor, no reduction in arsenic content was produced by washing the corneal specimens with saline solution, indicating that free lewisite no longer remained on the surface of the cornea after this interval.

Persistence of Arsenic and Toxic Material in the Cornea.—Two minutes after the instillation of 0.1 mg. of lewisite, about 4.6 micro-

TABLE 3.—*Toxicity of Corneal Juice After Instillation of 0.1 Mg. of Liquid Lewisite into a Rabbit Eye*

Time After Instillation of Lewisite	Maximum Ocular Reaction Produced by Intracorneal Injection of Corneal Juice into Normal Rabbit Eye
2 min.	47
30 min.	30
1 hr.	17
26 hr.	2
27 hr.	2
4 days	2 (two injections)

grams of arsenic was demonstrated within the corneal substance (table 2). In terms of lewisite, this is equivalent to about 10 per cent of the original dose applied. This amount of arsenic fell rapidly within the next thirty minutes to 1.4 micrograms, and after eleven hours only traces of arsenic were detected, and then irregularly. About 3 micrograms of arsenic was found within the cornea two minutes after the standard vapor burn was produced. The arsenic content was greatly diminished or absent after twenty-four hours.

Corneal juice obtained two minutes after production of the standard liquid burn caused a 47 per cent maximum ocular reaction when approximately one tenth of the juice was injected into a normal cornea (table 3). This toxicity progressively decreased in the course of thirty minutes and one hour and was absent at the end of twenty-six hours.

Rate of Penetration and Persistence of Arsenic and Toxic Material in the Aqueous.—Arsenic penetrated into the anterior chamber within one and one-half minutes after the application of 0.1 mg. of lewisite to the cornea and disappeared within thirty minutes after the exposure

(table 4). The aqueous was found to be toxic at the end of two minutes and ten minutes but had become nontoxic when tested at the end of thirty minutes (table 5).

Surface Decontamination After Exposure to Liquid Lewisite.—In view of the foregoing evidence that lewisite rapidly disappears from the surface of the cornea and penetrates into the cornea and aqueous, little or no therapeutic effect would be expected from the use of neutralizing agents which do not penetrate into the ocular tissues. This was found

TABLE 4.—*Microchemical Analysis of Arsenic in Aqueous Following Instillation of 0.1 Mg. of Lewisite on the Rabbit Cornea*

Time After Instillation of Lewisite	Amount of Arsenic, Micrograms
Aqueous control (no lewisite)	0.4 (blank of method = 0.0 to 0.5 microgram)
Needle control (for surface contamination in withdrawing aqueous)	0.2
1 min.	0.5
1.5 min.	2.2
5 min.	2.8
10 min.	3.2 (2 specimens)
20 min.	3.2
30 min.	0.0
1 hr.	0.0
2 hr.	0.5
11 hr.	0.9

TABLE 5.—*Toxicity of Aqueous After Instillation of 0.1 Mg. of Liquid Lewisite into a Rabbit Eye*

Time After Instillation of Lewisite	Maximum Ocular Reaction Produced by Intracorneal Injection of Aqueous into Normal Rabbit Eye
2 min.	47
5 min.	45
10 min.	32
30 min.	0
1 hr.	0
24 hr.	0
27 hr.	0
4 days	0

to be the case with three types of agents tried: (1) saline solution used in irrigation; (2) an iodine-containing solution, so-called Box I,[3] which inactivates lewisite in fifteen seconds in vitro, and (3) hydrogen peroxide (table 6).

3. This solution was devised by Dr. Leslie Hellerman as a method for the continuous regeneration of a limited low concentration of iodine:

Sodium iodide ..... 200 mg./100 cc.
Phosphate buffer p_H 7 ..... M/10
Sodium iodoxybenzoate in large excess
Solvent: 50% triacetin, 22% alcohol, 28% water

TREATMENT OF LEWISITE BURNS OF THE EYE WITH DIMERCAPROL

Since lewisite penetrates through the cornea into the aqueous within two minutes after exposure and produces irreversible changes in the tissues of the anterior ocular segment soon after ten minutes have elapsed, a successful decontaminating agent must have the capacity to penetrate rapidly and to compete successfully for both free and combined arsenic within the tissues. To obtain the maximum therapeutic effect from the local use of dimercaprol after exposure to lewisite, the following variables were studied: (1) local toxicity of dimercaprol for both the normal and the lewisite-burned eye; (2) optimum concentrations of dimercaprol and technics of application to the eye, (3) time limits during which treatment is efficacious and (4) species differences in toxicity and therapeutic effectiveness of dimercaprol.

Materials and Methods.—Both liquid and vapor burns were produced in the eyes of mongrel rabbits and liquid burns in the eyes of Macacus rhesus monkeys [4]

TABLE 6.—*Surface Decontamination of the Rabbit Eye Two Minutes After Instillation of 0.1 Mg. of Lewisite*

Number of Eyes	Decontaminating Agent	Maximum Ocular Reaction (%)	Final Corneal Opacity (%)	Days
3	100 cc. saline solution in irrigation.....	72% (S.D. ± 10)*	75% (S.D. = 19)	7
3	"Box I"—iodine solution [3].....	66% (S.D. = 3)	69% (S.D. = 2)	7
6	2% hydrogen peroxide (3 drops).....	69% (S.D. = 4)	65% (S.D. = 6)	7
3	Blood catalase alternating with 5% hydrogen peroxide every 5 sec. for 1 min.....	63% (S.D. = 20)	50% (S.D. = 30)	7
42	No treatment	74% (S.D. = 8)	69% (S.D. = 14)	7

* S.D. = standard deviation of the mean.

with lewisite containing 95 per cent of the active substance ($ClCH:CH.AsCl_2$). The liquid lewisite was loaded directly into a 0.25 cc. Luer tuberculin syringe. Because lewisite appeared to tarnish the stainless steel needles, a 26 gage platinum-iridium needle with a blunt point and gold-plated hilt was used, and care was taken to prevent moisture from precipitating the arsine oxide within the tip of the needle. The syringe was fitted with a micrometer in which one unit turn drove the plunger of the syringe down 1/200 inch (0.1 mm.), displacing a volume of 0.1 cu. mm. Droplets of this size were then put in contact with the cornea. Estimations of arsenic immediately following such an instillation on 10 rabbit corneas revealed that only 0.09 mg., or 0.05 cu. mm., of lewisite was actually delivered from the needle to the cornea—in other words, only 50 per cent of the theoretic quantity.

Rabbit eyes were exposed to saturated lewisite vapor at controlled temperatures by inserting the individual eye, proptosed between the lids, into the mouth of the Scholz vapor chamber.[5] All experiments were carried out with the animals

4. These experiments were performed on monkeys, which were made available to me by Dr. Cornelius P. Rhoads, Memorial Hospital, New York.

5. Scholz, R. O.: Personal communication to the author.

under ether anesthesia. The eyelids were closed within thirty seconds after exposure of the proptosed eye to either liquid or vapor lewisite.

The severity of the lesion produced was evaluated numerically by grading the important clinical symptoms elicited by the toxic agent (table 1). In order to obtain single values which could be used for statistical comparisons, the acuteness of the reaction was estimated by adding the maximum values for each symptom over the course of observation and converting the total to a percentage figure, the "maximum ocular reaction." An index of persistent corneal damage was obtained by adding the values of the corneal symptoms on the last day of observation, a total of 24 points or corneal perforation representing a 100 per cent lesion.

Preliminary experiments were devoted to the production of a reproducible and uniform standard lesion with lewisite, the technic and dosage to be used in treatment experiments. In general, it is necessary that the lesion be sufficiently severe to guarantee reproducibility, and yet an excessive dose should be avoided, which might mask the efficacy of a therapeutic agent of low potency. Such a threshold

TABLE 7.—*Severity of Lesions in Rabbit Eyes Following Standardized Exposure to Lewisite*

Type of Exposure	Number of Eyes	Maximum Ocular Reaction (%)	Final Corneal Opacity (%)	Number of Days Followed
0.1 mg. liquid lewisite.....	42	74 (S.D. = 8)*	69 (S.D. = 14)	7
	37	87 (S.D. = 8)	85 (S.D. = 19)	14
Exposed for 30 sec. to saturated vapor at 21-25 C.	18	70 (S.D. = 7)	63 (S.D. = 14)	7
	18	84 (S.D. = 10)	82 (S.D. = 14)	14

* S.D. = standard deviation of the mean.

lesion was produced by the instillation of 0.1 mg. of liquid lewisite either on the limbus or on the center of the cornea, usually resulting in perforation of the cornea within two weeks. A somewhat less severe, but consistent, lesion was produced by exposure of the eye to saturated lewisite vapor at 22 to 24 C. for thirty seconds. For use in treatment experiments, it was found that the optimum period of observation was seven days, since increased variability of the standard lesions was noted during the second week, probably as the result of secondary infection. The average values obtained for the standard lesions are given in table 7, together with the standard deviation of the mean.

Preliminary treatment experiments were made with a sample of dimercaprol ("BAL") obtained in the spring of 1942 from Dr. Peters' laboratory at Oxford. Little difference in toxicity or therapeutic efficacy could be made out between the British and the American sample. The American sample appeared to be slightly more toxic at higher concentrations and more therapeutic at lower concentrations, but the British sample was older at the time of comparison. Subsequent experience with later samples of American dimercaprol has shown results comparable to those reported here for the earlier sample.

Suitable solvents for dimercaprol were found in propylene glycol, ethylene glycol, redistilled thiodiethanol and triacetin. The substance is sparingly soluble and deteriorates rapidly in water but is effective therapeutically in aqueous solution. Because of many practical advantages in applying ointments to the eye, an oint-

ment containing dimercaprol was devised and prepared by Mr. Robert S. Fuqua, chief pharmacist of the Johns Hopkins Hospital, for use in these experiments.

	Percentage
Dimercaprol	5-10
Benzyl benzoate	5
Peanut oil	20
Absorbent base	20
Cetyl alcohol	10
Glycerin monostearate	14
Liquid petrolatum	21

This ointment was readily miscible with the tears of the eye and became semi-fluid within a minute after instillation. It was nontoxic for the normal rabbit and monkey eye and showed therapeutic efficacy comparable to that of dimercaprol in glycol solution.

Local Toxicity of Dimercaprol.—The tolerance of the normal cornea to dimercaprol was determined for rabbit and monkey eyes and partially for human eyes (table 8). The instillation of 0.1 cc. of a 50 or 35 per cent solution of dimercaprol in propylene glycol produced severe

TABLE 8.—*Tolerance of the Normal Eye to 0.1 Cc. of Various Solutions of Dimercaprol ("BAL")*

Species	Concentration of Dimercaprol, per Cent	Solvent	Maximum Ocular Reaction	Final Corneal Opacity	Days
Rabbit	50	Propylene glycol	67	67	7
	35	Propylene glycol	40	13	7
	25	Propylene glycol	20	0	2
	15	Propylene glycol	20	0	1
	10	Propylene glycol	5	0	0
	10	Ointment	2	0	0
	5	Propylene glycol	5	0	0
	3	Propylene glycol	5	0	0
	3	Water	2	0	0
Monkey	50	Propylene glycol	42	38	7
	35	Propylene glycol	37	21	6
	25	Propylene glycol	22	0	4
	15	Propylene glycol	0	0	0
	10	Propylene glycol	2	0	0
	10	Ointment	0	0	0
	5	Propylene glycol	0	0	0
Human—2 eyes	10	Propylene glycol	7	0	0
3 eyes	5	Propylene glycol	5	0	0

corneal damage in both rabbits and monkeys. This was characterized by a sheetlike opacification of the superficial layers of the cornea, which, if not unduly severe, sloughed off within a few days leaving clear stroma beneath. A 25 per cent solution of dimercaprol produced rather pronounced conjunctival reactions without corneal damage, and a 15 per cent solution produced a similar conjunctival reaction in rabbits but none in monkeys. A 10 per cent concentration or less caused no significant objective damage to the eye of the rabbit, monkey or man. The 10 per cent ointment and the 3 per cent aqueous solution of dimercaprol were nontoxic.

Since the development of an ocular lesion is almost completely prevented by the use of 2 drops of a 5 per cent solution of dimercaprol within two minutes after exposure to lewisite (see next section), the more severe reactions which follow the use of higher concentrations must be attributed to the toxic action of the therapeutic agent. This damage produced by dimercaprol can be distinguished clinically in an eye previously burned with lewisite. It is noteworthy that the reactions

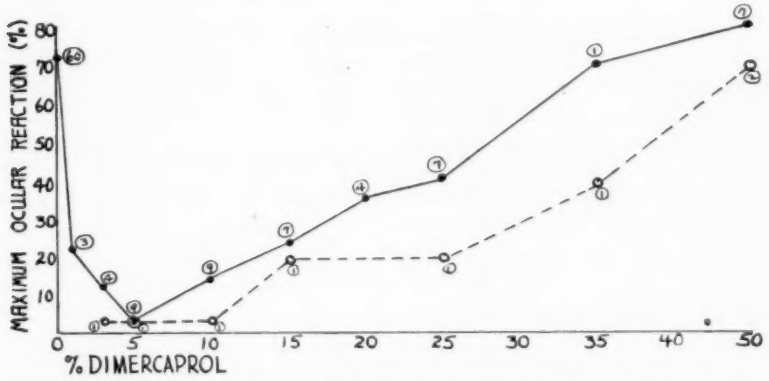

Fig. 3.—Effect of variations in the concentration of dimercaprol ("BAL") on reactions in normal rabbit eyes and in rabbit eyes burned with lewisite.

Values for eyes burned with either vapor or liquid lewisite and treated with 2 drops of a 5 per cent solution of dimercaprol in propylene glycol are indicated by the solid line and dots; values for normal eyes similarly treated, by the broken line and hollow circles. The numerals indicate the number of eyes tested.

TABLE 9.—*Effect of Variation in Concentration of Dimercaprol ("BAL") in Treatment of Lewisite Burns of the Rabbit Eye* *

Concentration of Dimercaprol Solution, per Cent	Number of Eyes	Maximum Ocular Reaction, per Cent	Final Corneal Opacity, per Cent	Number of Days of Follow-Up Observation
25	1	45	0	6
20	3	37	0	6
15	4	23	0	4
10	3	11	0	2
5	5	4	0	1
3	2	12	0	2
1	1	20	0	2
Untreated †	42	74 (S.D. = 8) ‡	69 (S.D. = 14)	7

* Instillation of 2 drops of dimercaprol in propylene glycol two minutes after application of 0.1 mg. of liquid lewisite.

† Instillation of 2 drops of propylene glycol had no influence on the severity of the lewisite lesion.

‡ S.D. = standard deviation of the mean.

produced by concentrations of dimercaprol of over 5 per cent in eyes previously exposed to lewisite are more severe than those produced by dimercaprol on the normal cornea (fig. 3). This suggests that lewisite lowers the tolerance of the cornea for dimercaprol.

Optimum Concentrations of Dimercaprol and Technics of Application in the Treatment of Lewisite Burns.—In the first of these experiments, standard burns produced with 0.1 mg. of liquid lewisite were treated two minutes later with 0.1 cc. of varying concentrations of dimercaprol in propylene glycol (table 9). Although all these concentrations, from 25 to 1 per cent, prevented the development of any corneal opacity lasting more than eight days, the use of 5 per cent solution also

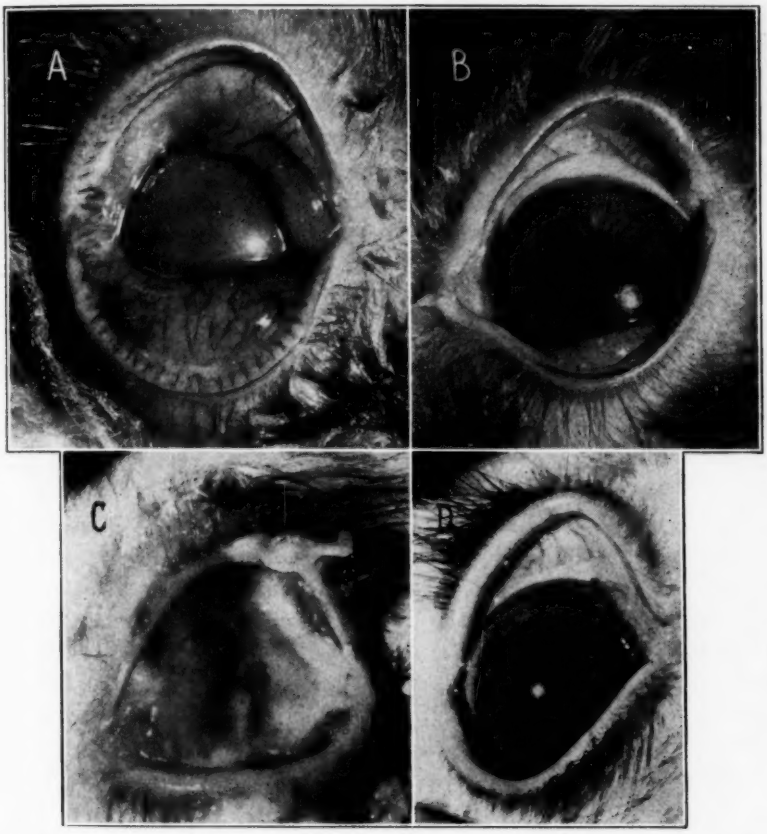

Fig. 4.—*A* and *B*, eyes exposed for thirty seconds to saturated lewisite vapor at a temperature of 22 C. twenty-four hours previously. The right eye (*B*) was treated two minutes after exposure with 0.1 cc. of a 5 per cent solution of dimercaprol ("BAL") in propylene glycol, and the left eye (*A*) was not treated. *C* and *D*, eyes exposed in a manner similar to that of the eyes shown in *A* and *B* four days previously and treated similarly.

prevented the development of any significant conjunctival reaction. Similar results were obtained in the treatment of vapor burns. There is, therefore, a moderately wide range of concentrations which produce spectacular cures of lewisite burns (fig. 4 *A* and *B*).

Experiments were carried out to determine the effect of larger doses or repeated instillations of various concentrations of dimercaprol. One instillation of 2 drops of solutions of 10 per cent concentration and over produced more reaction than the use of a single drop. The optimum time for the instillation of a second drop was after sixty minutes. Instillation of 1 drop of a 10 per cent solution of the British preparation every ten seconds for one minute, or every fifteen seconds for three minutes, produced severe corneal damage.

Two drops (0.1 cc.) of a 5 per cent solution of dimercaprol will theoretically neutralize about eighty times the standard dose of 0.1 mg. of liquid lewisite used in these experiments. However, the results of treatment following the instillation of 1.0 mg. of liquid lewisite were not nearly as good, probably because of the large area of instantaneous opacification produced by the acid component of the large lewisite droplet. The instillation of 2 drops of 10, 5 and 1 per cent solutions of dimercaprol two minutes after the instillation of 1.0 mg. of lewisite resulted in corneal opacities at the end of seven days of 67, 46 and 100 per cent, respectively. Similar treatment with 5 per cent dimercaprol of an eye exposed four times as long as that required for the standard vapor burn (i. e., two minutes) gave a maximum ocular reaction of 42 per cent and a final corneal opacity of 4 per cent at the end of seven days.

The use of 5 per cent dimercaprol ointment was found to be at least as effective as glycol solutions of the substance in the treatment of both liquid and vapor lewisite burns.

Time Limits During Which Dimercaprol Is Effective.—The instillation of dimercaprol in solution or ointment form within two minutes after the end of exposure to lewisite usually prevented the development of any significant conjunctival or corneal reaction. If treatment was delayed for five minutes, a transitory conjunctival and corneal reaction ensued, lasting a few days. Treatment ten minutes after exposure was definitely less effective, a mild to moderate corneal opacity persisting at the end of seven days. The use of dimercaprol within the first half-hour lessened the severity of the ocular lesion as compared with control eyes, but the damage to the eye was permanent.

These findings are in agreement with the observation that irreversible histologic changes are already detectable in the corneal stroma within ten minutes after exposure to lewisite and are fairly pronounced after thirty minutes. Also, at the end of thirty minutes, much of the arsenic has already disappeared from the tissues, and so the benefit derived from decontamination is diminished.

Use of Dimercaprol in Other Species.—Eleven eyes of monkeys were burned with liquid lewisite and 8 were treated with dimercaprol (table 10). The therapeutic efficacy and optimum concentrations of dimer-

caprol for monkeys were not significantly different from those found in the treatment of rabbit eyes.

Two drops of a 5 or 10 per cent solution of dimercaprol in propylene glycol was instilled into the eyes of 6 patients,[6] and 2 drops of propylene glycol alone was instilled into the eye of a seventh subject. Four patients had noninflamed blind eyes; 1 had low grade uveitis; 1 had a tuberculoma of the ciliary body which had eroded through the sclera, producing intense congestion externally, and 1 had bullous keratitis and glaucoma secondary to irradiation. In summary, it was found that the instillation of 0.1 cc. of a 5 or 10 per cent solution of dimercaprol in propylene glycol produced an immediate stinging sensation, at least partially due to the propylene glycol. In eyes not previously congested, blepharospasm prevented voluntary opening of the lids for about two minutes. The stinging sensation disappeared within five to thirty minutes. Pronounced lacrimation followed the instillation of the

TABLE 10.—*Treatment with Dimercaprol of Monkey Eyes Burned with 0.1 Mg. of Liquid Lewisite*

Dimercaprol Concentration, per Cent	Maximum Ocular Reaction, per Cent	Final Corneal Opacity, per Cent	Number of Days of Follow-Up Observation	Comment
15	32	29	6	0.1 cc. dimercaprol in propylene glycol applied 2 min. after exposure unless otherwise noted
10	5	0	2	Average for 3 eyes
10	0	0	1	Dimercaprol ointment used
5	8	0	3	Average for 2 eyes
3	10	0	3	.....
0	57	55	7	Average for 3 eyes

solution within a few seconds and disappeared in twenty to thirty minutes. Congestion of the conjunctival vessels with moderate edema of the conjunctiva developed within a minute or two and persisted for several hours. No evidence of damage to the normal cornea was detected by slit lamp examination and staining with fluorescein. In 2 eyes previously congested as a result of glaucoma and uveitis, respectively, severe and protracted ocular pain followed the instillation of solution of dimercaprol. A temporary exacerbation of the bullous keratitis occurred in 1 case. One patient with chronic noncongestive glaucoma and a second patient with glaucoma secondary to irradiation showed a reduction in intraocular tension of 12 mm. of mercury (Schiøtz tonometer) following the instillation of the drug. No subjective differences could be detected between the effect of the 5 and that of the 10 per cent solution.

6. These patients include 1 tested by Dr. Jonas S. Friedenwald and a second reported on by Dr. Cornelius P. Rhoads and Dr. Algernon S. Reese.

PERSISTENCE OF ARSENIC IN THE CORNEA AFTER THE APPLICATION OF DIMERCAPROL

The quantity of arsenic remaining in the cornea at stated intervals after a standard exposure to lewisite vapor was determined both for eyes treated with dimercaprol and for untreated controls (table 11). Whereas a trace of arsenic was demonstrated in the untreated corneas

TABLE 11.—*Influence of Dimercaprol on Rate of Disappearance of Arsenic from the Rabbit Cornea After Lewisite Vapor Burns*

Specimen Number	Dimercaprol	Time of Excision of Cornea	Amount of Arsenic, Micrograms
1	0	2 min.	2.7
2	..	.....	2.9
3	..	.....	3.3
4	..	30 min.	1.2
5	..	1 hr.	1.9
6	..	4 hr.	1.3
7	..	24 hr.	0.8
8	2 drops of 5 per cent	30 min.	0.0
9	dimercaprol solution	4 hr.	0.0
10	2 min. after exposure	24 hr.	0.0

after twenty-four hours, the corneas treated with 5 per cent dimercaprol showed no residual arsenic after thirty minutes. Thus, the use of dimercaprol facilitates the disappearance of arsenic from the tissues, probably by competing favorably with arsenic reversibly bound to the tissue components.

TABLE 12.—*Intravenous Treatment of Lewisite Burns of the Rabbit Eye with 15 per Cent Dimercaprol in Propylene Glycol*

Rabbit Number	Eyes	Weight of Rabbit, Kg.	Dose of Dimercaprol, Cc.	Time of Treatment, Min.	Maximum Ocular Reaction, per Cent	Final Corneal Opacity, per Cent	Days	Comment
1	.....	2.03	1.2	3	..	..	1	Rabbit died with convulsion
2	Right	1.8	0.5	2	37	0	7	Mild convulsions
	Left	....	...	..	47	25	7	
3	Right	2.0	0.4	15	47	6	7	Rabbit jittery
	Left	....	...	30	57	58	7	

INTRAVENOUS TREATMENT OF LEWISITE BURNS OF THE EYE WITH DIMERCAPROL

Toxic doses of English "BAL" (dimercaprol) were administered intravenously to 2 rabbits into whose eyes 0.1 mg. of lewisite had previously been instilled (table 12). The conjunctival, and to some extent the corneal, reaction in these 2 animals was less than the average ocular reaction in control animals.

SUMMARY AND CONCLUSIONS

Exposure of the eye to small quantities of lewisite liquid or vapor can produce a progressive lesion of the cornea, characterized by rapid tissue necrosis, pronounced edema and intense exudation. Lewisite is immediately hydrolyzed at the site of contact with the moist surface of the eye, liberating hydrochloric acid sufficient to produce a superficial corneal opacity. The more destructive characteristics of lewisite burns can be produced by a neutral solution of lewisite oxide containing the trivalent arsenic. Within two to four minutes after exposure to lewisite followed by closure of the lids, all toxic arsenical material disappears from the surface of the cornea, and within two minutes the drug can be demonstrated in the aqueous. Beginning ten minutes after exposure and becoming well marked in thirty minutes, irreversible histologic changes in the cornea can be detected.

A single instillation of 5 per cent dimercaprol solution or ointment from within two to five minutes after exposure to lewisite effectively prevents the development of serious ocular lesions. This excellent therapeutic effect of dimercaprol is due, in part at least, to its rapid penetration and withdrawal of toxic arsenical material from the tissues before irreversible histologic changes have developed.

OCULAR CHANGES IN THE BLOOD DYSCRASIAS

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IN CONSIDERING a group of diseases in which certain lesions in the eyes may develop, it is necessary to refer to features and conditions peculiar to these diseases which may influence to some extent the signs and symptoms that are likely to occur.

The blood dyscrasias include a very large group of disorders, with a variety of etiologic factors and with certain clinical manifestations. Moreover, there appears to exist a close relationship among many of these diseases, with only certain differences in their causation, which may determine the character of the blood picture, the clinical manifestations and the changes which may occur in the eyes. This is especially true of that part of the group designated as "the anemias."

For the purpose of simplicity, the more important diseases of the blood are here grouped according to the changes which occur in the individual constituents of the blood.

Diseases of Blood	Erythrocytes and Hemoglobin	Leukocytes	Platelets
1. Excess of red cells:			
Polycythemia, erythrocytosis	Increased; color index low	Increased	Increased
2. Anemias:			
Pernicious anemia	Decreased; color index high	Commonly decreased	Greatly diminished
Secondary	Decreased; color index low	Slightly increased	
Anemia of pregnancy	Increase in blood volume	Slightly increased	
Chlorosis	Decreased; color index low	Usually decreased	Unaffected or increased
3. Leukemias	Decreased	Increased, with number of abnormal cells	Decreased, with variable increase in chronic myelogenous form
4. Purpura hemorrhagica	Decreased	Slightly increased	Greatly decreased

EXCESS OF RED CELLS

Polycythemia.—This condition has been described by Whitby and Britton [1] as an excess of cells in the blood. They classified the disease as primary and secondary polycythemia, the primary form being known as erythremia and the secondary form as erythrocytosis. In the primary form, there are an "unexplained hyperplasia of the erythron with polycythemia, an increased blood volume and often an increase in the number of capillary blood vessels." In the secondary form, there are hyper-

Presented at a meeting of the New York Academy of Medicine, Section of Ophthalmology, Nov. 19, 1945.

1. Whitby, L. E. H., and Britton, C. J. C.: Disorders of the Blood, ed. 4, Philadelphia, The Blakiston Company, 1942.

trophy of the erythron and polycythemia as a compensatory reaction. The blood is thereby given an increased oxygen-carrying capacity in cases in which tissue oxygenation is imperfectly performed by a normal blood.

Cohen,[2] in a review of the lesions of the fundus complicating polycythemia, with a report of cases, pointed out that while the general manifestations of the two forms are similar, except that cyanosis is likely to be more intense in the secondary form, mild or severe ocular manifestations may be associated with either type.

The following case is here presented as an example of the secondary form of polycythemia, or erythrocytosis, which resulted from a toxic

Fig. 1.—Fundus (left) in a case of erythrocytosis.

process; the patient had sudden impairment of vision in the left eye, with a hemorrhage and edema in the macular region.

H. S., aged 37, was employed as a supervisor in a zinc-refining plant. He first came under observation Feb. 12, 1945 because of rather sudden impairment of vision in the left eye, with a resulting acuity of 20/100. There was a small paracentral scotoma in the left eye. External examination revealed practically a normal condition. Ophthalmoscopic examination of the left eye showed that the media were clear; the optic nerve head was somewhat blurred in outline. Just above the macula was a small, apparently recent, superficial hemorrhage, which was accompanied with an area of edema extending downward toward the macula. The retinal veins were distended and bluish, while the arteries had a somewhat bluish purple appearance. No other lesions were observed. Examination of the right eye revealed no hemorrhages or other pathologic change except a distention of the retinal veins and a bluish color of both arteries and veins.

2. Cohen, M.: Lesions of the Fundus in Polycythemia: Report of Cases, Arch. Ophth. **17**:811 (May) 1937.

Examination of the nose, throat, sinuses and teeth showed nothing abnormal. Examination by Dr. Benjamin Gouley revealed that the heart was normal and the electrocardiogram not remarkable. On fluoroscopic examination the heart appeared to be of approximately upper normal size. The pulsations were regular and of fair amplitude, and there was no evidence of pulmonary congestive failure. The urine showed a trace of albumin. The prostate gland was normal. The Wassermann reaction was negative. The blood pressure was 130 systolic and 90 diastolic. There was no evidence of cardiac failure. The liver was moderately enlarged, the lower border being palpable 3 fingerbreadths below the right costal margin in the midclavicular line. The lower pole of the spleen was barely palpable. The blood count showed the presence of polycythemia. The hemoglobin concentration was 106 per cent; the red cell count was 5,400,000 and the white cell count 14,400, with 74 per cent polymorphonuclear leukocytes, 25 per cent lymphocytes and 1 per cent monocytes. There was no increase of blood platelets. The differential count showed no abnormality of the white blood cells. The red cells, however, presented two interesting changes, in contrast to each other, namely, (1) the presence of full-bodied erythrocytes in large numbers and (2) the presence of many erythrocytes undergoing hemolysis, leaving narrow rims of many cells. This was interpreted by the hematologist as indicating that the erythrocytes were being destroyed while, at the same time, the bone marrow was overproducing. The hemoglobin later rose to 130 per cent, with 6,000,000 red cells. The white cell count was 11,000. The blood smear showed subsidence of peculiar destructive changes previously noted in the erythrocytes, and toxic hypoplastic anemia, although expected, did not occur. The urobilinogen level was normal. There was a slight, subclinical increase in the icterus index—10. The blood urea nitrogen was normal—14 mg. per hundred cubic centimeters—as were the other chemical constituents of the blood. The hemoglobin was now reduced to 102 per cent and the white cell count to 8,000.

The patient was hospitalized and remained under treatment, with gradual improvement in his ocular and general condition. It is interesting to note that this man, who had no previous complaints and was in apparent good health, came under observation because of the visual disturbance and that the condition of the blood was discovered when he was referred for a complete physical examination. In the opinion of the hematologist, the polycythemia was an expression of a toxic process, probably a chemical intoxication, which was responsible at the same time for the intraocular hemorrhage. The toxic stimulus, however, was not severe enough to produce anemia. The enlargement of the liver was part of the process, rather than a complication of cardiac dysfunction.

The hemorrhage and edema in the left eye gradually cleared, and vision returned to normal with a correcting lens. When last seen (April 3, 1945) the patient had resumed his employment and was using a respirator mask in the plant.

THE ANEMIAS

The anemias as a group probably include the largest number of diseases of the blood, with a variety of etiologic factors, which probably have best been classified by Ottenberg.[3] According to this classification, the anemias which are of greatest interest to the ophthalmologist are

3. Ottenberg, R.: Reclassification of the Anemias, J. A. M. A. **100**:1303 (April 29) 1933.

those which result from the following conditions: (1) deficiencies: (*a*) iron deficiency (loss of blood and hypochromic anemia); (*b*) deficiency of the antianemic principle, as in pernicious anemia and pernicious anemia of pregnancy; (2) injury to the blood-forming organs: (*a*) osteosclerosis, leukemias, Hodgkin's disease, Gaucher's disease, Niemann-Pick disease, Schüller-Christian disease and other disorders with lipid deposits in the bone marrow; (*b*) interference with regeneration of blood: diseases of the spleen, such as Banti's syndrome.

Most of the chronic forms of disease with disturbances or alterations in the hemopoietic system exhibit a marked progressive anemia, enlargement of the liver and spleen and a distinctive blood picture. The variety and extent of the ocular changes may be said to depend in general on the type and extent of involvement of the other structures, particularly those of the blood and the blood-forming tissues.

The ocular changes associated with the anemias include chiefly: (1) changes in the appearance and size of the retinal vessels, (2) changes in the retina itself (principally pallor, hemorrhages and edema), (3) edema and swelling of the optic disks and (4) exophthalmos. The first three are characteristic especially of the primary and secondary anemias (pernicious anemia, sudden loss of blood, purpura, the leukemias and splenic anemia). Exophthalmos and displacement of the eyeball occur chiefly with lymphoma, Hodgkin's disease, lymphosarcoma and chloroma, and also with the diseases involving the bone marrow with lipid deposits in the bones of the orbit, as in Schüller-Christian disease. (In other diseases, such as Gaucher's disease and Niemann-Pick disease, which may be accompanied with mild hypochromic anemia, the ocular changes are produced by the characteristic deposits in other structures.)

The following tabulation is a brief summary of a case of pernicious anemia (hyperchromic anemia), in which large hemorrhages were present in the retina but none was observed with the so-called white center.

Pernicious Anemia (Hyperchromic Anemia)

Mr. A. G.: Age 55. Occupation: Baker. Weight: 154 lb. (69.9 Kg.). Admitted 10/13/45

Blood Picture	Clinical Signs	Ocular Changes
Red cells: 1,890,000 Hemoglobin: 6.9 Gm.—48% White cells: 5,100 Platelets: 190,000 Sternal puncture: Many megaloblasts; appearance of marked hyperchromic macrocytic anemia, compatible with diagnosis of pernicious anemia or hyperchromic anemia of other origin. Blood pressure: 110/60 Blood sugar: 120 mg /100 cc. Urea nitrogen: 18 mg./100 cc.	Precordial pain; dyspnea; slight distention of abdomen; pallor of skin; spleen and liver not palpable; achlorhydria; digestive disturbances; edema of ankles	Optic disks round, with margins slightly blurred and slight edema around disk; retinal veins distended; arteries pale and tortuous; a few small, linear retinal hemorrhages in both eyes; fairly large hemorrhages along both superior and inferior temporal vessels, none with white center

In cases in which the changes in the blood occur suddenly, as, for example, from severe trauma or after sudden, severe hemorrhage, changes in the retina with visual loss may follow.

A number of cases with retinal hemorrhage, exudates and loss of vision from trauma to other parts of the body have been published in the literature from time to time. Recently, Rados, Bedell and Spaeth [4] each described a case of Purtscher's disease (angiopathy of the retina) resulting from trauma. The injury in most cases is of a crushing variety, which results in venous congestion with secondary manifestations in the cranium and changes in the retina. The latter are characterized by a retinal angiopathy, the principal features of which are engorgement of the retinal veins, retinal hemorrhages and the presence of edema and white spots in the retina. Papilledema, papillitis and large preretinal hemorrhages may also occur. Both eyes are usually involved. Vision may not be greatly impaired unless the macula is involved. The lesions may heal, leaving no trace of their former presence. The

Fig. 2.—Fundi in a case of hyperchromic macrocytic anemia (pernicious anemia).

retinal hemorrhages may be numerous and are usually of the striate, punctate or petechial variety. They lie contiguous to the vessels but are apparently not related to the white spots.

The white spots constitute the prominent and interesting feature. They appear as bright white specks, predominantly between the disk and the macula. They frequently obscure the veins, to which they show a close relation, and have been described as resembling scattered "snowflakes" in outline and appearance. Their margins are indistinct and

4. Rados, A.: Lymphorrhagia Retinae Traumatica, Arch. Ophth. **6**:93 (July) 1931. Bedell, A. J.: Traumatic Retinal Angiopathy, ibid. **22**:351 (Sept.) 1939. Spaeth, E. B.: Traumatic Liporrhagia Retinalis (Verhoeff), Purtscher's Disease, ibid. **31**:191 (March) 1944.

are split into fine striations. They regress rapidly, diminish in number and density and dissolve into fine, silvery bright puncta and striae. The exact cause of these spots is still undetermined. It had been considered that a thrombosis of the intravascular sinuses occurs or that a sudden increase of intracranial pressure takes place, cerebrospinal fluid being forced along the vaginal sheath to produce the retinal changes. Spaeth cited Verhoeff, who stated that it is possible for fat droplets to be generally diffused in the retina, together with the presence of a generalized hemorrhagic situation in the retina, the brain or both, as a result of factors other than the emboli themselves. The name "traumatic liporrhagia retinalis" was suggested as adequately describing the condition. The appearance of the spots strongly suggested the presence of fat, and they were said to occur only where there had been fracture and obstruction of the retinal veins. They are most likely due to venous obstruction associated with traumatic hyperlipemia. Distention of the small vessels might permit the fat to pass into the tissues, either by diapedesis or through small breaks in the distended vessels. Usually some blood is extravasated with the fat.

Severe loss of blood from injury or profuse hemorrhage for any reason which results in severe anemia may be followed by temporary or permanent impairment of vision. Cox [5] reported a case in which loss of vision followed a crushing injury to the chest. The loss of vision was attributed to anemia following massive loss of blood.

Langdon [6] reported a case of amaurosis following severe uterine hemorrhage which lasted for long periods at a time. Examination of the eyes revealed only a "white fluffy material" covering the disks and obscuring their margins. There were no intraocular hemorrhages. The blood count showed 45 per cent hemoglobin, 1,880,000 red cells and 22,000 white cells. After treatment for several months, the disks were described as slightly pale, but otherwise the fundi were normal. Vision improved to nearly normal in each eye.

Edema of the nerve heads and choked disks have been observed in patients with different types of blood dyscrasia. A few cases of purpura hemorrhagica have been reported with choked disk in the absence of an intracranial pathologic process. The mechanism for the production of papilledema and choked disk in these cases is therefore not clear. Three such cases of thrombopenic purpura with bilateral choked optic disk and a fourth case in which choked optic disks appeared in the course of recurrent severe hemorrhages from a gastric ulcer were

5. Cox, R. A.: Amblyopia Resulting from Hemorrhage, Arch. Ophth. **32:** 368 (Nov.) 1944.

6. Langdon, H. M.: Amaurosis After Uterine Hemorrhage with Restoration of Vision Following Transfusion, Arch. Ophth. **10:**99 (July) 1933.

reported by Watkins, Wagener and Brown.[7] It was believed that no intrameningeal or intracerebral bleeding had occurred in any of the cases and that some other factor was responsible for the occurrence of the papilledema. They suggested that the mechanism of production was "a local reaction of the tissues of the optic nerves to anoxemia that resulted from loss of blood." This conclusion was based principally on the need of an adequate supply of oxygen by the retina and optic nerve to insure normal function, which is rapidly and permanently lost in even short periods of deprivation of oxygen. The reduced oxygen-carrying power of the blood resulting from acute or chronic loss of blood might produce anoxemia of the tissues of the optic nerve and retina sufficient to induce papilledema and edema of the retina. It would occur for the same reason in cases of angiospastic retinitis. The appearance of edema of the optic nerve and retina is often observed in cases of severe anemia from any cause.

LEUKEMIAS

Anemia, usually of a progressive type, is an almost constant feature of the leukemias. Although typical leukemic changes in the eye are described under the designation of "leukemic retinitis," the ocular manifestations may show variations, and only part of the usual picture may be observed, especially in the early stages. The most constant, and probably the earliest, evidence in the eyes in leukemia are the changes in the appearance, caliber and tortuosity of the veins, which become dilated, tortuous and darker than normal. A pinkish yellow color of the fundus, the appearance of some edema of the optic disks and retinal hemorrhages are the other important changes.

Gibson [8] in a report of the retinal changes in 22 cases of leukemia, attached great significance to these changes in the retinal veins, since they are nearly always present, even when hemorrhages are not seen. His observations showed that the changes in the retinal veins, with the presence of the retinal hemorrhages, is so characteristic of leukemia as to point to the diagnosis. This sign is even enhanced when a narrow white line is observed on each side of the vein, indicating perivenous infiltration.

The significant ocular changes in the leukemias were also described by Goldbach,[9] who attributed the changes in the caliber and tortuosity of the veins to the altered blood picture. He stated that edema of

7. Watkins, C. H.; Wagener, H. P., and Brown, R. W.: Cerebral Symptoms Accompanied by Choked Optic Discs in Types of Blood Dyscrasia, Am. J. Ophth. **24**:1374 (Dec.) 1941.

8. Gibson, G. G.: Clinical Significance of the Retinal Changes in Leukemia, Arch. Ophth. **20**:364 (Sept.) 1938.

9. Goldbach, L. J.: Leukemic Retinitis, Arch. Ophth. **10**:808 (Dec.) 1933.

the retina is produced secondarily to the retarded blood flow, especially in the veins and capillaries, which results in seepage of the blood elements from the capillaries with secondary pressure on the perivascular lymph sheaths. The yellow to light pink color of the fundus in leukemic retinitis was attributed to the degree of infiltration of the choroid, the condition of the retinal circulation and the type and location of the hemorrhages. The lymphatic obstruction, retarded circulation, dilated and tortuous veins, extravasation of plasma and relatively unyielding lamina cribrosa tend toward local edema and congestion of the disk.

The size and shape of the hemorrhages may vary from small, narrow streaks to massive areas in the more pronounced cases. The typical hemorrhage of leukemic retinitis, with its so-called white center, is not always in evidence. It is also true, as was stated by Goldbach, that the hemorrhages are not characteristic of any of the various types of the disease. The amount of hemorrhage in the retina also seems to be proportional to the degree of anemia and bears no relationship to the number of white cells. This is in accord with the observations of both Goldbach and Gibson.

The summaries of 4 cases are here presented. They include 2 cases of chronic myelogenous leukemia, 1 case of subacute lymphatic leukemia (aleukemic phase) and 1 case of osteosclerosis with aleukemic leukemia, including a summary of the blood picture, clinical signs, ocular changes and autopsy observations in 2 cases. One of the 2 cases of chronic myelogenous leukemia is of interest because of the fact that although the anemia was comparatively moderate a number of retinal hemorrhages with moderate edema and distended veins were present. The second case was that of a woman aged 27 who died after an illness of more than two years. She had anemia of about 50 per cent, but the ocular changes during the terminal stages of the disease consisted only of moderate distention of the retinal veins, with no retinal hemorrhages in either eye.

CASE 1.—*Chronic myelogenous leukemia*

Mrs. C. di A.: Age 47. Height: 5 ft. 3 in. (167 cm.), Weight: 213 lb. (96.6 Kg.).
Admitted 12/26/44. Discharged 2/1/45

Blood Picture	Clinical Signs	Ocular Changes
Red blood cells: 3,190,000-3,220,000 Hemoglobin: 9.2 Gm.—64% 9.8 Gm.—68% 9.5 Gm.—66% 12/26/45 2/1/45 White blood cells: 250,000 110,000 Roentgen therapy—January 1945 9/5/45 9/18/45 White blood cells: 50,000 — 80,000 Red blood cells and hemoglobin unchanged	Patient complained of poor vision Splenomegaly, hepatomegaly, distended abdomen Weight, 256 lb. in Aug. 1944 Blood pressure, 114/78 Blood sugar, 90 mg./100 cc. Wassermann and Kahn reactions of blood negative	Optic nerve heads blurred, surrounded with edema; retinal veins engorged and tortuous; superficial retinal hemorrhages and large preretinal hemorrhage with yellowish center in left eye

CASE 2.—*Chronic myelogenous anemia*

Mrs. J. C.: Age 27. Blood pressure: 130/90. Four previous admissions
Admitted 5/22/45. Died 8/2/45

Blood Picture				Autopsy (1 Hr. After Death)	Ocular Changes
	5/22	6/4	7/30	Chronic myelogenous leukemia with leukemic infiltration of all organs Spleen: 2,840 Gm. Liver: 3,160 Gm. Blood: Wassermann reaction negative; Rh negative	No typical evidences of leukemia except moderate congestion of central retinal veins
Red blood cells	2,750,000	Transfusion	2,400,000		
Hemoglobin	8.1 Gm. 56%	10.5 Gm. 70%	7.25 Gm. 50%		
White blood cells	43,000	70,000	60,000		
6/7/45: White cells, 4,900 after intravenous injection of 10 cc. of radioactive phosphorus (strength, 10 millicuries) Myeloblasts 76-80% at death					

The patient with subacute lymphatic leukemia, a 19 year old student, had marked anemia, and examination of the eyes showed edema of the nerve heads, distended retinal veins and a number of preretinal hemorrhages in both eyes. The hemoglobin concentration ranged from 22 to 30 per cent, and the red cells, from 1,200,000 to 1,700,000.

CASE 3.—*Subacute lymphatic leukemia (aleukemic phase)*

A. M.: Student, Age 19. Admitted 10/7/40. Died 12/7/40

Blood Picture	Clinical Signs and Autopsy Observations	Ocular Changes
Hemoglobin: 4.9-3.1 Gm. 34%-27% Red blood cells: 1,740,000-1,200,000 White blood cells: 1,300-2,900 Platelets 80,000 Coagulation time 5½ min. Bleeding time 11 min. Sternal puncture: Marrow counts support diagnosis of subacute lymphatic leukemia in aleukemic phase (counts unchanged until death) Blood pressure: 115/58 Blood sugar: 104 mg./100 cc. Urea nitrogen: 112 mg./100 cc. Wassermann and Kahn reactions negative	Weakness, pallor; cervical, axillary and inguinal lymph adenopathy Autopsy: Spleen. Hyperplasia Liver: Cloudy swelling; amyloid degeneration; fatty change; congestion; edema Bone marrow: Congestion, fibrosis, leukemic cell infiltration	Margins of disks fairly well outlined in both eyes; retinal veins congested; four or five preretinal hemorrhages in each eye; one smaller retinal hemorrhage with white center in right eye

The patient with osteosclerosis and aleukemic leukemia, a man aged 51, complained of weakness, vertigo and swelling of the abdomen and ankles. He was jaundiced; the liver and spleen were greatly enlarged, and there was a large mass on the right side which was thought to be the liver. Roentgenographic examination revealed diffuse osteosclerosis of the bones of the spine, shoulder girdle and long bones. A subacute hyperplastic lymphadenitis was present. There was an anemia of somewhat less than 50 per cent. The eyes showed anemia of the retina and edema of the optic disks. The retinal veins were distended, but no hemorrhages were observed.

CASE 4.—*Osteosclerosis with aleukemic leukemia*

Mr. D. L.: Age 51. Admitted 6/16/45. Discharged 8/14/45

Blood Picture	Clinical Signs and Biopsy Observations	Ocular Changes
Red blood cells: 2,430,000-2,810,000 Hemoglobin: 50-54% White blood cells: 5,800-10,500 Blood sugar: 109 mg./100 cc. Urea nitrogen: 27.8 mg./100 cc. Wassermann reaction negative	Liver and spleen enlarged; all bones of chest, dorsal part of spine and long bones showed evidences of osteosclerosis Biopsy of inguinal lymph nodes showed hyperplastic lymphadenitis	Blurred nerve heads with moderate edema in retina around disks; congested veins, arteries pale; no hemorrhages or exudates

BANTI'S SYNDROME

This disease is characterized by an enlarged spleen, a tendency toward gastric hemorrhage; epistaxis; cirrhosis of the liver with ascites, especially in the later stages, and progressive anemia. Although the cause of the condition is obscure, it is known that venous congestion of the spleen takes place, associated with cellular proliferation. The anemia is also unexplained except that direct loss of blood from hemorrhage may be a factor. The early anemia of the disease may be due to splenic dysfunction, without any evidence of a direct destructive action by the spleen or of any definite toxic or hemolytic agent. Hypochlorhydria or achlorhydria usually occurs.

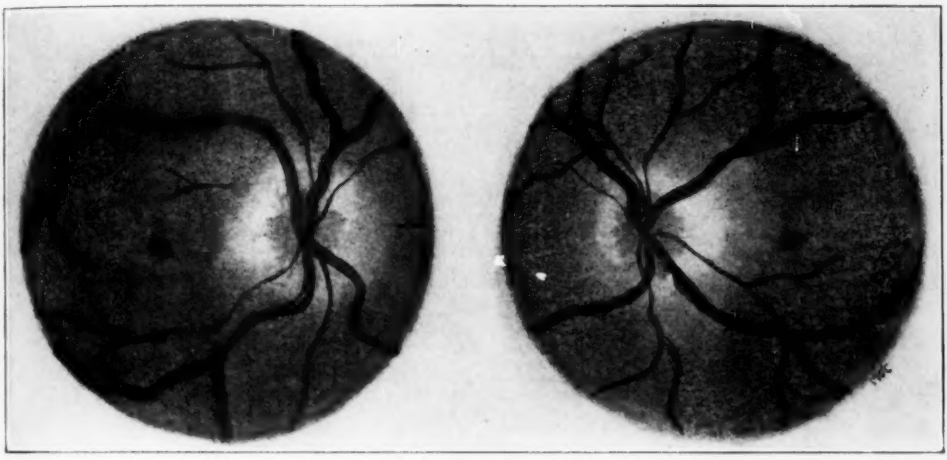

Fig. 3.—Fundi in a case of anemia associated with osteosclerosis and aleukemic leukemia.

The following case of Banti's syndrome with autopsy is of particular interest because of the pronounced changes in both eyes and because no case of this disease has been found in the literature in which ocular manifestations are described.

W. B., a 4 year old boy, had been ill for two years under the care of Dr. Eugene Rush, at Mount Sinai Hospital, Philadelphia. The ocular changes were observed during the last week of the disease.

The clinical signs and symptoms included early loss of weight, pallor of the skin, cachexia and moderate jaundice. Gastric hemorrhages and epistaxis occurred both early and late in the disease. The abdomen was greatly distended with ascites, and abdominal paracentesis was performed several times. The liver and spleen were greatly enlarged.

Examinations of the blood revealed hemoglobin values of 48, 50, 56 and 32 per cent. The red cell count was 2,000,000. There was a prothrombin deficiency of 28 per cent.

Ophthalmic Examination.—The sockets were deep, and there was a pale conjunctiva with moderate jaundice in both eyes but no other external abnormalities of note. Ophthalmoscopic examination revealed that the media were clear in each eye. The optic disks were blurred in outline and showed considerable edema. The retinal veins were greatly distended, and the arteries were pale. There were also a marked pallor and moderate edema of both retinas. A number of round and linear, superficial hemorrhages were seen in the retina of each eye close to ths disk and also in the periphery. Many of these hemorrhages surrounded a white center and were similar to those which occur in leukemic retinitis. In the left eye there were also a number of small, yellowish white areas of infiltration around the macula. Since Banti's syndrome is not a distinct entity and there are no specific pathologic changes which identify the disease, the changes in the optic nerves, retinas and retinal vessels in this case can be attributed to the severe anemia which was present.

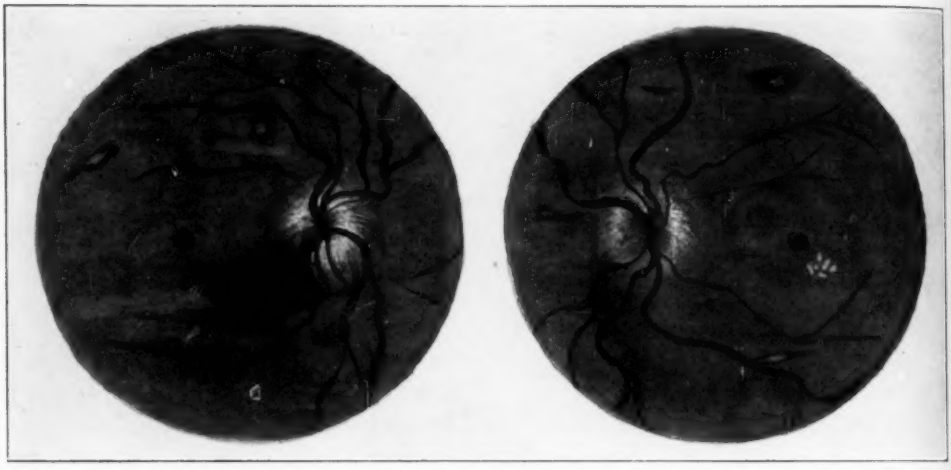

Fig. 4.—Fundi in a case of Banti's syndrome (splenic anemia) with severe anemia.

Autopsy, by Dr. David Meranze, pathologist, Mount Sinai Hospital, confirmed the clinical diagnosis. The appearance of the spleen was compatible with Banti's syndrome. There were cirrhosis of the liver, marked hemorrhage into the pulmonary alveoli and bilateral pleural effusion.

OTHER BLOOD DYSCRASIAS

Exophthalmos is probably the most prominent clinical sign in cases in which lymphoma occurs in the orbit. The following types of lymphomas were described by Leinfelder and O'Brien[10]: (1) sclerosing (Hodgkin's disease), (2) endothelial (lymphoendothelioma), (3) lymphoblastic (lymphosarcoma) and (4) lymphocytic (lymphatic leukemia). These lesions are closely related and are characterized by hyperplasia and metaplasia of the lymphoid and blood-

10. Leinfelder, P. J., and O'Brien, C. S.: Lymphoma of the Eye and Adnexa: Report of Thirteen Cases, Arch. Ophth. **14**:183 (Aug.) 1935.

forming tissues, with enlargement of the lymph nodes, spleen and liver and changes in the bone marrow. The blood picture is typical only in lymphatic leukemia, whereas the white cells are usually normal in the other types. In the latter, however, secondary anemia occurs, especially in the later stages of the disease. Except in lymphatic leukemia, the ocular changes in this group are rare. Nodular thickening of the lids with lymphomatous infiltration of the eyes and lids and unilateral or bilateral exophthalmos may occur. Leinfelder and O'Brien [10] observed exophthalmos as a result of lymphomatous infiltration of the orbit in 6 cases.

A case of lymphoma of the orbits which was considered to be associated with chronic lymphatic leukemia was reported by Cohen [11] in a man aged 65 with bilateral exophthalmos. The laboratory diagnosis of leukemic growth was made after removal of the growth.

Chloroma is a rare condition characterized by the presence of a neoplastic tumor of the orbit, with exophthalmos probably the most prominent ocular finding. The tumors are typically green in color and occur in association with involvement of the hemopoietic tissues and a leukemic blood picture. A case occurring in a man aged 55 with bilateral exophthalmos was reported by Gump, Hester and Lohr.[12] In this case the blood picture was one of monocytic leukemia and pathologic involvement of the reticuloendothelial system. Another case of the disease occurring in a 4 year old boy with exophthalmos of the left eye was reported by Frost,[13] who stated that the underlying pathologic process reveals that the disease is a particular type or an unusual form of myelogenous leukemia.

The prominent ocular manifestation in Schüller-Christian-Hand disease is exophthalmos, usually bilateral. The other characteristics of this syndrome are degeneration and lipid deposits in the bones, especially the bones of the skull and orbit, and diabetes insipidus. The disease is congenital and familial in origin and is peculiar to male children, and the pathologic changes involve the reticuloendothelial system. The liver, spleen, lymph nodes and kidney are moderately enlarged. A severe anemia occurs, with extensive involvement of the bone marrow.

SUMMARY

The blood dyscrasias are a group of diseases characterized by various alterations in the circulating cellular constituents, which accompany abnormal processes in other tissues.

11. Cohen, M.: Orbital Lymphoma in Chronic Lymphatic Leukemia: Report of a Case, Arch. Ophth. **11**:617 (April) 1934.

12. Gump, M. E.; Hester, E. G., and Lohr, O. W.: Monocytic Chloroma, Arch. Ophth. **16**:931 (Dec.) 1936.

13. Frost, A. D.: Chloroma: Report of a Case with Hematologic Study, Tr. Am. Acad. Ophth. **42**:123, 1937.

A case of erythrocytosis with ocular changes, which was probably toxic in origin and was of temporary duration, is described.

The anemias are classified, and those diseases with ocular changes in which the anemia is an important factor are described. A case of hyperchromic anemia (pernicious anemia) with ocular changes is presented.

The ocular changes in the leukemias are described, with reference to the literature. Four cases of leukemia with their ocular changes are summarized.

A case of Banti's syndrome (splenic anemia) with profound anemia and marked changes in the retinas and both optic nerves is presented. These ocular changes were probably the result of the anemia.

CONCLUSIONS

Most of the diseases included among the blood dyscrasias are accompanied with anemia of various types and degrees. The ocular changes occurring in these diseases may involve principally the optic disk, retinal vessels and retina in the form of edema, hemorrhages and venous distention. The degree of anemia present may in general be said to determine the type and extent of the ocular involvement.

Exophthalmos is the prominent ocular manifestation in those diseases in which the abnormal process occurs in the orbit or involves the bones of the orbit.

DISCUSSION

DR. PAUL REZNIKOFF, New York: It is not generally realized that patients do not go to a hematologist directly—they are sent to him by another physician. A blood dyscrasia is usually suspected by the ophthalmologist, the dentist or the dermatologist, or by an internist occasionally; but the patient practically never says, "I have something wrong with my blood so I am going to a hematologist." Therefore it is important for specialists, such as ophthalmologists, to recognize conditions which may be due to a hematologic condition. This brings me to the point which hematologists are always stressing, that the hematologic findings in cases such as those presented by Dr. Tassman must be correlated with the entire condition of the patient, as he has done. In a general way, there is no such thing as a primary blood disease. All disorders of the blood are secondary. That is why hematologists have dropped the classification of primary and secondary anemias. The so-called pernicious anemia is not primary, for it is due to a deficiency in a nutritional factor. Such a blood dyscrasia as leukemia, for example, is considered primary because the cause is not known. If it were known, the disease would not be called primary.

Sulfonamide therapy has introduced many hematologic conditions, some of which are fatal, and the same is true of thiouracil, which is used for hyperthyroidism and has produced several cases of agranulocytosis. Fatal cases of purpura may result from gold salt therapy in the treatment of arthritis.

One type of anemia which I did not see in Dr. Tassman's classification, and one which is important for the ophthalmologist, is hemolytic anemia. This frequently results in hemorrhages in the retina; surprisingly, often the first evidence of hemolytic anemia is found by the ophthalmologist, who notes jaundice of the sclera, occasionally missed by the clinician, especially if his office has artificial illumination.

As one may gather from Dr. Tassman's report, with the possible exception of leukemia, there is practically no blood disease for which the changes in the eye are pathognomonic—so that one can say that the patient has polycythemia, or so-called Banti's syndrome, or anemia. What can be gathered from the ophthalmic examination is that something may be amiss with the bleeding or clotting mechanism; and in the further investigation of these factors a hematologic study is indicated.

I do not know of any case of hemophilia in which a so-called nontraumatic, or spontaneous, hemorrhage involved the eye. If such a patient is struck in the eye, he will bleed; but few hemophilic patients have had "spontaneous" hemorrhages. That is not true of hemorrhages in other sites. For the normal patient, the trauma is of minimal significance, and he repairs the damage rapidly; but the hemophilic patient cannot. I suspect from the fact that there are so few reports of so-called spontaneous hemorrhage in orbital structures that trauma caused by the ordinary movement of the eyeball is rare. The eye is well protected from trauma, such as occurs to the knee or other joints. Even in the urinary tract the passage of urine produces some trauma, and hemophilic patients may bleed profusely from the urinary tract.

One of the problems encountered with polycythemic patients, and one not yet solved, is hemorrhage in the retina, which causes a great deal of disturbance. However, it is generally considered that patients with this disease have a rather sluggish circulation, which is conducive to thrombosis of the veins; they also have a high red blood cell count, so that the plasma has little room for clot retraction.

My final remark has to do with hemorrhages in the hematologic conditions, which Gibson has indicated may be due to anemia. That is puzzling to hematologists, because in even the severest anemias hemorrhage is not so frequent as in a condition like thrombopenic purpura, with little anemia, or in leukemia, with only moderate anemia. It is my impression that if Gibson could repeat his studies, and correlate hemorrhage not only with the white blood cell count and anemia but with the platelet count as well, he might find that most of the hemorrhagic conditions in the eye are due to thrombopenia. Hemorrhage is very common in leukemia, especially of the acute type, and in the so-called aplastic anemia; and, as can be seen from Dr. Tassman's tables, it is not uncommon to have some degree of thrombopenia in pernicious anemia, as well as in so-called Banti's syndrome.

DR. RUDOLF AEBLI, New York: In my own experience in the last two years I have had 2 cases in which expulsive choroidal hemorrhage followed cataract extraction. Subsequently, I found that both the patients had polycythemia vera.

OCULAR SPOROTRICHOSIS

Report of a Case

DAN M. GORDON, M.D.
NEW YORK

SPOROTRICHOSIS is a widespread but relatively uncommon disease.

> . . . The condition is not figured by Smith in his Atlas of Skin Diseases in the Tropics, nor does Loewenthal mention it in his account of the diseases of the Skin in negroes.[1]

Most of the reported cases have come from France and the United States. Isolated cases have been reported in Germany, Switzerland, Austria, Belgium, England, Scotland, China, Turkey, Italy, Spain, Madagascar, where it is rather common, and South America. Three large series of cases have been described among South African miners.[2]

Du Toit [2b] stated that only 206 cases had been reported in the American literature up to January 1940. Link in 1809, cited by Moore and Kile,[3] described the genus Sporotrichum chiefly as a saprophyte on wood. Sporotrichum badium was the first species described and illustrated. The first pathogenic species was probably that described by Montagne, in 1886. Schenck [4] in 1898 described the first case, in which a typical lesion occurred on a finger, followed by the formation of a chain of nodules. This observer did the first scholarly work on sporotrichosis. He recovered the organism twice in cultures. The organism was classified by E. F. Smith, of the United States Department of

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Read in part at a meeting of the New York Academy of Medicine, Section of Ophthalmology, May 21, 1945. A brief abstract of this paper, with discussion, appeared in the November-December 1945 issue of the ARCHIVES, page 463.

1. Dangerfield, L. H., and Gear, G.: Sporotrichosis Among Miners on the Witwatersrand Gold Mines, South African M. J. **15**:128, 1941.

2. (*a*) Dangerfield and Gear.[1] (*b*) du Toit, C. J.: Sporotrichosis on the Witwatersrand, Proc. Transvaal Mine M. Officers A. **22**:111, 1942. (*c*) Pijper, A., and Pullinger, B. D.: An Outbreak of Sporotrichosis Among South African Native Miners, Lancet **2**:914, 1927.

3. Moore, M., and Kile, R. L.: Generalized, Subcutaneous, Gummatous, Ulcerating Sporotrichosis, Arch. Dermat. & Syph. **31**:672 (May) 1935.

4. Schenck, B. T.: On Refractory Subcutaneous Abscesses Caused by a Fungus, Possibly Related to the Sporotricha, Bull. Johns Hopkins Hosp. **9**:286, 1898.

Agriculture, as belonging to the genus Sporotrichum, which had been created by Link in 1809. Mount [5] stated:

> Great credit is due Schenck, for as a result of his research there was added to dermatology the description of a disease worked out completely in the United States.

Brayton in 1899 described a similar case but failed to prove the diagnosis by culture.[6] Hektoen and Perkins [6] described their case in 1900 and isolated the organism in culture. Hektoen gave the name Sporotrichum schencki to the fungus. The first non-American case was described by de Beurmann and Ramond [7] in 1903. In 1905 the organism was named Sporotrichum beurmanni by Matruchot and Ramond, cited by du Toit,[2b] who isolated and studied the fungus.

It was once thought that there was a difference between Sporotrichum schenckii, which had been described in the United States, and Sporotrichum beurmanni, described by the French. Davis [8] proved that the two were identical, with Schenck getting credit as the first observer. It would seem, then, that it would be more correct historically and as a matter of custom to refer to the organism as Sporotrichum schenckii. Actually, the literature contains both terms, to the confusion of the reader.

In 1907 Danlos and Blanc [9] published the first case in which sporotrichosis had been known to involve the eye or its adnexa. Here the lid was involved. The same year de Beurmann, Gougerot and Laroche,[10] whose names are so firmly linked to the history of sporotrichosis, also cited a case of involvement of the lid. Greco [11] reported a similar case from the Argentine in the same year.

In 1908, 2 cases were reported from France—a case of infection of the lid, by Morax and Carlotti,[12] and a case of orbitopalpebral involve-

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5. Mount, L. B.: Sporotrichosis, with Report of a Rather Unusual Case, Arch. Dermat. & Syph. **25**:528 (March) 1932.

6. Hektoen, L., and Perkins, C. F.: Refractory Subcutaneous Abscesses Caused by Sporothrix Schencki, J. Exper. Med. **5**:77, 1900.

7. de Beurmann and Ramond: Abcès sous-cutanés multiples d'origine mycosique, Ann. de dermat. et syph. **4**:678, 1903.

8. Davis, D. J.: Interagglutination Experiments with Various Strains of Sporothrix, J. Infect. Dis. **12**:140, 1913; The Formation of Chlamydospores in Sporothrix Schenckii, ibid. **15**:483, 1914; The Effect of Potassium Iodid on Experimental Sporotrichosis, ibid. **25**:124, 1919.

9. Danlos and Blanc: Un cas de sporotrichose palpébrale, Bull. et mém. Soc. méd. d. hôp. de Paris **24**:1450, 1907.

10. de Beurmann; Gougerot, and Laroche: Gomme de la paupière, Bull. et mém. Soc. méd. d. hôp. de Paris **27**:1046, 1907.

11. Greco, O.: Sporotrichose linfangitica nodular vegetante, cited by Bedell.[35]

12. Morax, V., and Carlotti, P.: La sporotrichose palpébrale, Ann. d'ocul. **139**:418, 1908.

ment, by Fage.[13] In 1909, 6 cases appeared in the French literature. Thibierge and Gastinel [14] cited a case of involvement of the lid. Morax [15] had a case of conjunctival infection. Fava [16] reported on a laboratory infection of his own conjunctiva. He accidentally got an emulsion of the organisms into his eye. One-half hour later he irrigated the eye with a 1 : 4,000 solution of mercury oxycyanide. Eleven days later he noted a foreign body sensation in the eye and observed a yellow spot on the tarsal conjunctiva. The preauricular and postauricular nodes became enlarged, and a growth of the organism was obtained on culture. The condition cleared up with oral use of potassium iodide over a period of two and a half months. In Burnier's [17] and Weill's cases the cul-de-sac was involved. In Bonnet's [18] case the infection was orbitopalpebral. De Beurmann and Gougerot [19] cited a fatal case of generalized sporotrichosis with destruction of one eye. The same case was also reported by Lagoutte and Briau.[20]

Gifford Sr.[21] reported the first American case in 1910, and, according to him, the first in the world in which the conjunctiva was the seat of the infection. However, Morax had reported a similar case the year before. According to Gifford, he had seen 5 other cases of a condition which clinically appeared to be sporotrichosis, but he had been unable to recover the organism.

In 1910, 5 more cases occurred in France. In Gougerot and Dubosc's [22] case the lid was involved. Morax and Cruchaudeau [23] cited a case in which the conjunctiva was invaded. Velter [24] reported a case of orbital abscess. The patient refused treatment with potassium iodide, and the lesion consequently did not heal while he was under

13. Fage, A.: Sur un cas de sporotrichose, Progrès méd. **24**:248, 1908.

14. Thibierge, G., and Gastinel, P.: Trois cas de sporotrichose dermohypodermique, Bull. et mém. Soc. méd. d. hôp. de Paris **26**:537, 1909.

15. Morax, V.: La sporotrichose de l'appareil visuel, Ann. d'ocul. **141**:321, 1909.

16. Fava, A.: Un cas de sporotrichose conjonctivale et palpébrale primitives, Ann. d'ocul. **141**:338, 1909.

17. Burnier: Un case de sporotrichose gommeuse hypodermique ulcéreuse disséminée localisations conjonctivales, Ann. d'ocul. **141**:344, 1909.

18. Bonnet: Sporotrichose, Lyon chir. **2**: 515, 1909.

19. de Beurmann and Gougerot: Sporotrichose cachectisante mortelle, Bull. et mém. Soc. méd. d. hôp. de Paris **26**:1046, 1909.

20. Lagoutte and Briau, cited by de Beurmann and Gougerot.[19]

21. Gifford, H.: Sporotrichosis of the Eye-Ball and Eyelids, Ophth. Rec. **19**:573, 1910.

22. Gougerot and Dubosc: Sporotrichose palpébrale, cited by Toulant.[60]

23. Morax, V., and Cruchaudeau: Sporotrichose conjonctivale primitive, Ann. d'ocul. **144**:69, 1910.

24. Velter, E.: Un cas de sporotrichose orbito-palpébrale primitive, Ann. d'ocul. **144**:65, 1910.

Velter's observation. Danlos and Flandin [25] reported a case of de Lapersonne's [26] of disseminated sportrichosis with resultant phthisis bulbi. Jeanselme and Poulard [27] similarly reported a case of generalized sporotrichosis with involvement of the iris. The patient lost his sight as a result of the exudate formed.

Four more cases were published in France in 1911. Chaillous [28] reported a case of conjunctival infection which occurred after handling a horse with suppurating eyes. Morax [29] told of a case in which the lacrimal sac was involved with fistula formation. This patient refused treatment with potassium iodide, and there was no healing while he was under observation. Legry and associates [30] noted a case of disseminated sporotrichosis which resulted in iridocyclitis. This condition responded to iodides. Dor's [31] case was one of corneal involvement in an infant with generalized sporotrichosis following vaccination for smallpox. In this case, also, the condition responded to potassium iodide.

In 1912 de Lapersonne [26] described a case of generalized sporotrichosis in which iridocyclitis and resultant phthisis bulbi ensued. The condition responded to potassium iodide, although the eye had to be removed as a result of pain. The fungus was not identified in the eye, although it was observed in other tissues. Chaillous [28] the same year reported a case of disseminated sporotrichosis with intraocular involvement and perforation of the sclera. The eye was lost, although the rest of the lesions responded to potassium iodide.

In 1913 Morax [32] reported a case of involvement of the lid which simulated a lacrimal fistula.

In 1914, 5 cases were reported. Morax[33] reported a case of uveitis which was due to sporotrichosis, without other lesions. The organism was recovered in a culture of the aspirated pus. Thibierge and

25. Danlos and Flandin, C.: Sur un cas de sporotrichose traité et meconnu pendant 2 ans, Bull. et mém. Soc. méd. d. hôp. de Paris **29**:206, 1910.

26. de Lapersonne, F.: Sporotrichose oculaire, Presse méd. **20**:93, 1912.

27. Jeanselme, E., and Poulard: Sporotrichose de l'iris, Ann. d'ocul. **144**:65, 1910.

28. Chaillous: Sporotrichose conjonctivale primitive, Ann. d'ocul. **145**:47, 1911.

29. Morax, V.: Sporotrichose primitive du sac lacrymal, Ann. d'ocul. **145**:49, 1911.

30. Legry, Soudel and Velter: Sporotrichose gommeuse disséminée avec lésions oculaires (iridocyclite et gommes de l'iris) et spina ventosa sporotrichosique, Bull. et mém. Soc. méd. d. hôp. de Paris **32**:124, 1911.

31. Dor, L.: Un cas de sporotrichose oculaire, Clin. opht. **17**:121, 1911.

32. Morax, V.: Sporotrichose primitive des paupières simulant une fistule lacrymale, Ann. d'ocul. **149**:183, 1913.

33. Morax, V.: Uvéite sporotrichosique avec gomme sporotrichosique épisclérale sécondaire, Ann. d'ocul. **152**:273, 1914.

Chaillous,[34] in France, cited a case of intraocular involvement, and the first case in which the retina was affected. There were nodules in the subcutaneous tissue and on the lid, as well as iritis. Three cases were reported from the United States in the same year. Bedell [35] reported a case of chronic conjunctival sporotrichosis in which six concretions were removed from the lacrimal sac. When these were cultured, after being crushed, the organism was recovered. Wilder and McCullough,[36] earlier that year, reported a case of the disease occurring in one of them (C. P. M.), in which a pure culture of Sporotrichum schencki was accidentally spilled into the conjunctival sac. The patient had pronounced constitutional symptoms, as well as pain and lymphangitis, but the infection responded to potassium iodide. In their discussion of this case, Dwyer and Larkin [37] mentioned a case of their own, never officially reported, in which an infection of the lid occurred secondary to an ulcer of the finger.

In 1917 Leoz [38] reported the first Spanish case, which he had seen in 1915. The patient had corneal scars and ulcers with associated submaxillary adenitis. The organism was recovered in the culture. He injected a 5 per cent solution of potassium iodide into the nodules, with good results. This author stated that before he recognized sporotrichosis he had seen a condition which in retrospect he knew to be sporotrichosis but which at the time he had called syphilis. The patient had died within two months of the diagnosis.

Oreste [39] reported a case of conjunctival involvement in 1919. No cultures were made in this case, and hence the diagnosis was not confirmed. The patient responded to potassium iodide.

In 1920 Aloin and Vallin [40] published a case of involvement and perforation of the frontal bone, together with an abscess of the upper lid. This condition also responded to potassium iodide.

In 1921 Gonzalez [41] reported an interesting case in which the disease had gone on for four years, with orbital involvement. He

34. Thibierge and Chaillous: Sporotrichose palpébrale, Clin. opht. **20**:126, 1914.

35. Bedell, A. J.: A Case of Chronic Sporotrichosis of the Eye, Tr. Am. Ophth. Soc. **13**:720, 1914.

36. Wilder, W. H., and McCullough, C. P.: Sporotrichosis of the Eye, J. A. M. A. **62**:1156 (April 11) 1914.

37. Dwyer and Larkin, in discussion on Wilder, W. H.: Ocular Sporotrichosis, abstracted, Arch. Ophth. **45**:162, 1914.

38. Leoz, G.: Esporotricosis ocular, Arch. de oftal. hispano-am. **17**:293, 1917.

39. Oreste, A.: Sporotricosi congiuntivale, Gazz. med. napol. **2**:326, 1919.

40. Aloin, H., and Vallin, H. G.: Ostéite perforante du frontal par sporotrichose, Lyon méd. **129**:859, 1920.

41. Gonzalez, J. de J.: Mycotic Tumor of Orbit, abstracted, Am. J. Ophth. **5**:244, 1922.

finally obtained a smear showing the organism, and with potassium iodide therapy even the exophthalmos cleared. No culture was made here.

In 1922 Sobly Bey,[42] of Egypt, reported a case which he thought was one of sporotrichosis of the inner canthus. However, the cultural characteristics of the organism differed from those of S. schenckii. The patient was cured with potassium iodide.

In 1922 Sanford Gifford [43] reported a case of involvement of the lacrimal sac with fistula formation and infection of the ethmoid sinuses. The patient required an intranasal exenteration of the ethmoid cells, with local applications of trichloroacetic acid, administration of potassium iodide and local use of iodine, before the condition cleared up. This was the first true case of dacryocystitis due to Sporotrichum since Morax' case, reported in 1911.[29] Gifford noted 2 other cases in which sporotrichosis was suspected but in which the cultures were negative.

In 1924 Sanford Gifford [44] reported a case of infection of the lid which responded to potassium iodide and local application of iodine. It is significant that the name Gifford is associated with 3 of the 9 American cases reported prior to this paper.

In 1927 King [45] cited a case of generalized sporotrichosis in which a lid was secondarily involved. This patient responded slowly to treatment with potassium iodide until large doses were given intravenously, combined with roentgen irradiation.

In 1928 Alvis [46] reported the eighth American case, that of a patient who had previously had trachoma, of eight years' duration. He noted a linear excoriation of the conjunctiva, filled with a cheesy material. Scrapings of this, when cultured, yielded a growth of the organism.

In 1930 Hill [47] reported the ninth American case, and the last one published in this country prior to the present case. Hill's case was a most interesting one. The onset was with a small nodule on the upper lid, which another physician had opened, with a diagnosis of chalazion. It grew over a period of five years, gradually involving the globe, with formation of a hypopyon ulcer and involvement of the malar and frontal bones. Radical emptying of the orbit and removal of an antral wall had to be resorted to. Three months later a small

42. Sobly Bey, M.: A Case of Palpebral Sporotrichosis, Tr. Ophth. Soc. U. Kingdom **42**:388, 1922.

43. Gifford, S.: Ocular Sporotrichosis, Arch. Ophth. **51**:540 (Nov.) 1922.

44. Gifford, S. R.: Further Note on Ocular Sporotrichosis, Arch. Ophth. **53**: 264, 1924.

45. King, H.: Sporotrichosis with Report of an Unusual Case, South. M. J. **20**:541, 1927.

46. Alvis, B. Y.: Sporotrichosis of Eyelid, Am. J. Ophth. **11**:381, 1928.

47. Hill, H. F.: Sporothrix Infection of the Eye and Adnexa, Tr. Am. Acad. Ophth. **35**:128, 1930.

nodule was removed from the canine fossa. The patient was reported as cured five months later. This is the second American case in which the lesions failed to respond to iodides alone.

In 1930 Amias [48] reported a case of sporotrichosis of the bulbar conjunctiva. The condition occurred in a young boy who had scratched his eye with a chestnut twig. This was followed by the development of a punched-out ulcer and associated lymphangitis, as well as constitutional symptoms. The organism was recovered in culture, and the lad responded to treatment with potassium iodide over a period of two months.

In 1931 Fonseca [49] reported a case of sporotrichosis of the lower palpebral border. Here, again, the organism was recovered in the culture, and potassium iodide produced a cure in three months.

In 1936 Fazakas,[50] of Germany, reported a case of sporotrichosis occurring in the lacrimal canal of a patient who had had the lacrimal sac removed nine years before. Potassium iodide produced a cure here.

In 1937 Cavallacci [51] cited the case of a man who had been struck in the left eye with a willow branch. Fifteen days later a small gray spot developed at the limbus. This gradually grew, although vision was not affected. The culture revealed the true diagnosis, and treatment with iodides locally and orally effected a cure within one month.

In 1939 Mendes de Castro and Amato [52] cited a case with involvement of the cornea.

In 1940 Decoud and Schujman [53] reported a case of involvement of the lid, which responded to potassium iodine.

In 1941 Dangerfield and Gear [1] reported on 74 cases of sporotrichosis occurring in miners in South Africa. In 2 of these cases the infection was primary on the lid and responded to iodides.

In 1943 Nino [54] cited a case of mycotic ulcer of the cornea which he thought was due to sporotrichosis.

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48. Amias, V.: Un caso de esporotricosis de la conjunctiva bulbar, Arch. de oftal. hispano-am. **30**:644, 1930; abstracted, Am. J. Ophth. **14**:845, 1931.

49. Fonseca, A.: Oculopalpebral Sporotrichosis, Rev. de oftal. de São Paulo **1**:33, 1931; abstracted, Am. J. Ophth. **15**:479, 1932.

50. Fazakas, A.: Sporotrichose des unteren Tränenkanälchens, Klin. Monatsbl. f. Augenh. **96**:227, 1936.

51. Cavallacci, G.: Granuloma del limbus da Sporotrichum, Arch. di ottal. **44**:247, 1937; abstracted, Arch. Ophth. **20**:660 (Oct.) 1938.

52. Mendes de Castro, M., and Amato, G. G.: Sporotricose: Uma localização interessante, Gaz. clin. **37**:306, 1939.

53. Decoud, A. C., and Schujman, S.: Esporotricosis palpebral, An. argent. de oftal. **1**: 422, 1940; abstracted, Am. J. Ophth. **24**:1332, 1941.

54. Nino, F. L.: Úlcera micótica de córnea: Estudio micológico de una observación, Prensa méd. argent. **30**:797, 1943.

In this same year Bolanos and Trejos [55] reported 2 cases of multiple facial lesions, which included the lids and which responded to iodides.

In 1942 du Toit [2b] reported on 650 cases of sporotrichosis occurring among 2,500 employees in a mine in South Africa. Although this author gave an excellent review of the subject of sporotrichosis, he failed to analyze his cases from the point of view of anatomic localization. Nor do any of his 37 pictures indicate lesions of the eye or its adnexa. He did state that "practically the only type seen on the face" occurred as a warty growth on the forehead between the eyes.

REPORT OF CASE

Mrs. M. H., a housewife aged 48, came to the eye clinic of the New York Hospital on May 1, 1944, complaining of a "sore" on her left upper eyelid. She stated that she had had this for about two weeks but could give no definite history of trauma. There were pronounced swelling and redness of the lid, with a granulomatous ulcer in the center. The ulcer was covered with a crust, under which there was considerable purulent material. The skin adjacent to the lesion was fixed. Vision was 20/15 in each eye. Examination of the eyes themselves and of the lacrimal apparatus revealed nothing pathologic. General physical examination also showed an essentially normal condition.

On the third day there was a hard nodular swelling on the lid just temporal to the ulcer. On the fifth day the reaction at the site of the original lesion was still intense, but there were now two small, pea-sized nodules temporal to the ulcer. One of these was just at the external canthus. By the ninth day enlargement of several of the nodules draining the left upper lid was noted. The skin overlying theses nodules was red and indurated. An agar culture of material taken at this time was later reported to show no growth.

The pateint returned again on May 12, by which time sporotrichosis was suspected. Further questioning elicited the statement that she had been working in her garden, among poinsettias, about a week prior to the onset of the "stye" on her lid. She could recall wiping the back of her soiled hand across the left supraorbital region. A culture on Sabouraud's medium was made by Dr. George Lewis of material taken from one of the nodules by aspiration. This was reported (by Miss Mary Hopper) to show a growth of S. schenckii five days later. At the same time an intracutaneous test with sporotrichin was performed by Dr. M. B. Sulzberger. At this visit the skin over the nodules was more inflamed than before. The nodules were of a hard, rubbery consistency and extended in a chain toward the left ear. The node at the external canthus was now becoming larger, softer and somewhat heart shaped. There was a palpable preauricular lymph node.

Two days later the intracutaneous test gave a positive reaction, with a papule 5 by 5 mm. in size, surrounded by an area of erythema measuring 22 by 30 mm. The patient stated that the reaction had been much greater on the day before.

On the sixteenth day another culture on Sabouraud's medium was made by Dr. Lewis and Miss Hopper; this, too, was reported to show a growth of S. schenckii four days later. At this visit the patient complained of some pain in the left supraclavicular region, and a postauricular node was palpable.

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55. Bolanos, L., and Trejos, A.: Múltiples coincidencias en dos casos de esporotricosis facial, Rev. méd. Costa Rica **5**:369, 1943.

Treatment with a saturated solution of potassium iodide was started on the twentieth day of the disease. My colleagues and I had purposely not started this medication sooner, as we were anxious to secure at least one positive culture before so doing. The intial dose was 10 drops four times daily. This was gradually increased to 15, then to 20 and by the thirty-first day to 25 drops four times daily.

Five days after the institution of the potassium iodide therapy the initial lesion had begun to involute, but the nodules had now become painful and the overlying skin angry looking. The pain radiated down the left arm. The pre-

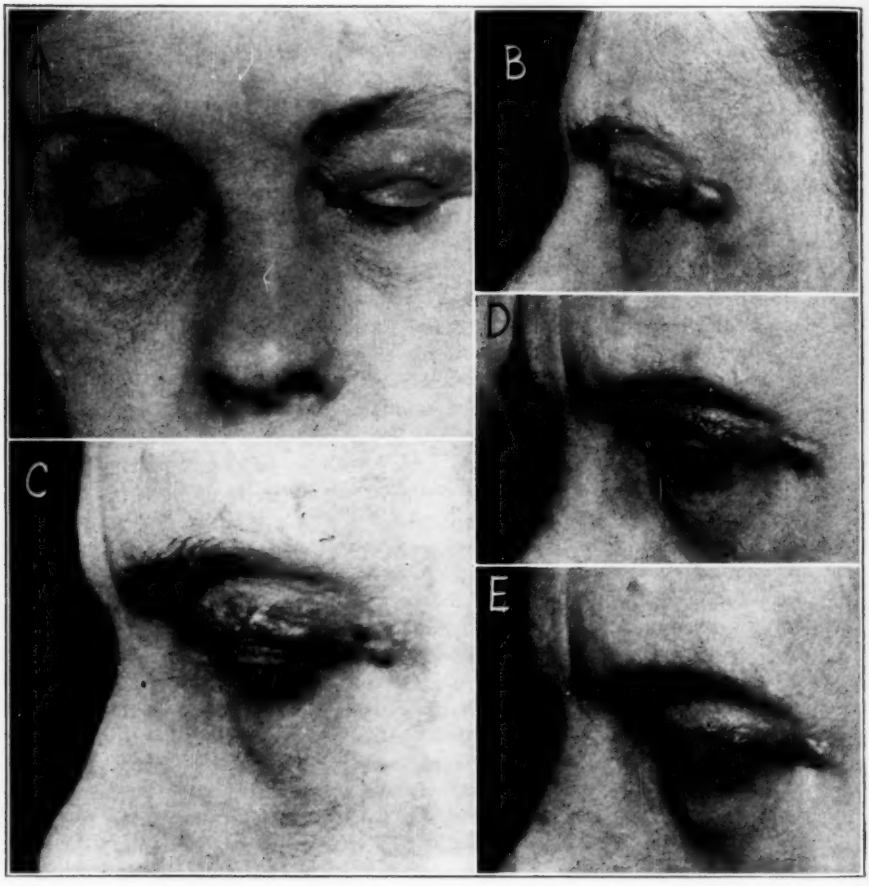

Fig. 1.—*A*, sporotrichosis of the left upper lid as seen about the twenty-sixth day of the disease. *B*, patient about the thirty-ninth day of the disease. Note lesions on the upper lid, with a heart-shaped lesion at the external canthus. Treatment with potassium iodide had been started five days earlier, with an increase in the reaction. *C*, patient eleven days after iodide therapy had been started. Note beginning involution of the lesion. *D*, appearance approximately five weeks after beginning of iodide therapy. The lesions on the lid were markedly involuted. *E*, patient two months after iodide therapy had been started. The lesions no longer contained sporotricha and were almost flat.

Fig. 2.—Kodachrome showing appearance of the left upper lid, with typical lesions.

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sumption is that there were deeper nodes in the supraclavicular area, although these were not palpable. The patient also complained of headache. There was no rise in temperature.

Eleven days after iodide therapy was started there was pronounced improvement. All the lesions about the lid were smaller, as was the preauricular node. The postauricular node was slightly increased in size.

My colleagues and I next saw the patient six weeks after institution of the medication. She had discontinued taking the iodide one week previously because of its bad taste. We insisted that she resume taking the drug for at least another two weeks. The lesion on the lid was almost completely involuted. The one at the external canthus was still soft. A culture of material taken from it by aspiration was reported to show no growth.

The patient was last seen by us on Dec. 7, 1945, at which time she showed a small healed scar on the left upper lid and one at the external canthus.

ETIOLOGIC FACTORS

Sporotrichosis is a mycosis caused by a parasite, the sporotrichum. Several members of the genus have been described, but it is generally agreed that S. schenckii is the only important member in this country as seen clinically. Many of the sporotricha occur as saprophytes and are found on vegetation in various parts of the world. They may also be found in the excreta of human and animal carriers. The fungus has been found in horses, dogs, rabbits, cats and rats. It has been discovered in the mouths and on the fur of healthy rats and on the coats of horses which had the disease. It is thought that the infection can occur as the result of rat bite.

The most common history is one of injury with a thorn, cactus or brier preceding the onset of the disease, and in many of the cases reported in the literature the patients have been florists [56] or people working around gardens. In 3 of the largest series reported, those from the South African mines, the occurrence was on the basis of rock and other mining injuries. In these series about 50 per cent of the primary lesions were at the site of injury. There have not been any cases reported in the literature of the disease being spread by contact between human beings. Moore and Manting [57] reported a case which was thought to be due to a mosquito bite, with lodging of the fungus in the skin at the site of the bite.

It is of interest that du Toit [2b] successfully isolated a sporotrichum from dust floating about in the air and from timber during one of the South African epidemics. However, this author was not certain that the organism which he found was the one responsible for the disease

56. Gastineau, F. M.; Spolyar, L. W., and Haynes, E.: Sporotrichosis: Report of Six Cases Among Florists, J. A. M. A. **117**:1074 (Sept. 27) 1941.

57. Moore, M., and Manting, G.: Sporotrichosis Following Mosquito Bite. Arch. Dermat. & Syph. **48**:525 (Nov.) 1943.

among the workers, as it was less virulent when rats were inoculated and failed to produce clinical symptoms in human volunteers. This same author also stated that the "saprophytic existence of the fungus is known to reduce its virulence." Hence spontaneous reductions in the outbreaks tend to occur.

CLINICAL APPEARANCE

Clinically, sporotrichosis is seen as a chronic infection which is characterized by the formation of nodules in the lymph nodes, epidermis or subcutaneous tissues. These nodules tend to occur in chainlike formation and break down to form indolent ulcers. The initial lesion is commonly referred to as the chancre. While the infection is commonly found about the fingers, there have been reported in this country, prior to this paper, 7 cases of primary involvement of the eyes. The lesions of sporotrichosis are destructive; and, as has been pointed out earlier in this paper, fatalities have occurred.

About the eye, the disease most commonly manifests itself as ulcers of the lids and conjunctiva. These tend to begin as large subcutaneous gummas and break down to form ulcers. The latter are deep and may even develop in the bony walls of the orbit.

The globe itself can be implicated in three ways: by direct trauma, by direct extension or by the blood stream.

Lewis and Hopper [58] summarized the various clinical types as follows:

1. Localized lymphangitic type. Most of the South African and American cases fall under this head. Here, an initial lesion occurs on some exposed part of the body. This appears as a hard, round, movable and painless nodule, over which the skin can be moved. The nodule later becomes indurated and attached to the skin, which gradually changes in color from pink to purple and later becomes black and necrotic as the nodule softens and breaks down. While this lesion may, rarely, remain as the sole sign of the disease, it is not typical. Usually the initial lesion is followed in a week or more by a painless ascending inflammation in the regional lymphatics. In their course a chain of secondary subcutaneous nodules may be seen. The lymphatics do not typically become enlarged, as do the nodes in tularemia or in any of the disorders commonly included under the term Parinaud's conjunctivitis. Systemic symptoms and generalized involvement are uncommon, as is spontaneous recovery. The involuting lesions leave residual scars of varying degrees. The incubation period is about one week but may vary from three days to three weeks.

58. Lewis, G. M., and Hopper, M. E.: An Introduction to Medical Mycology, Chicago, The Year Book Publishers, Inc., 1939.

2. Disseminated subcutaneous type. This form is more commonly described in cases from France and is not characteristically associated with an initial chancre. Usually small, hard, painless nodules are found in the subcutaneous tissues. Within a period of several weeks the overlying skin becomes involved, the nodules undergoing central softening and abscess formation. The latter may leave indolent ulcers, with firm, indurated borders. If untreated, new lesions continue to appear.

3. Disseminated ulcerating type. This type is much like the second form except for its tendency to earlier ulceration. Du Toit stated that "many clinicians still tend to think only of the lymphangitic type, forgetting that the ulcerative and other forms are seen."

4. Epidermal type. As I have previously stated, the initial lesion is usually subcutaneous. In this type the epidermis at distant sites becomes secondarily involved. Rare cases of pure epidermal disease have been described. It is stated that the organism may remain as a saprophyte in the mouth or on the mucous surfaces after the disease is apparently cured, rendering the patient a possible carrier.

5. Systemic type. Here, the deeper tissues and viscera are implicated. This is most commonly seen in cases of the disseminated type in which early treatment has not been given. The skeletal system may be involved here, as may the inside of the globe.

6. Allergic lesions. Sporotrichids, similar to trichophytids, have been described by de Beurmann.

Most American cases have been of the lymphatic type, whereas most of the French cases have been of the "multiple ulcerative type." The nodules and the granulomatous ulcers are apparently not necessarily directly connected, and either may appear on any part of the body. Du Toit, however, stated that the lesions have never been reported on the feet or over the pelvic girdle.

The ophthalmologist must bear in mind the fact that the lymphatic type is not the only one seen in this disease but that the ulcerative and other forms are commonly met with. These tend to appear as small granulomatous ulcers, which are covered with a crust and beneath which an irregular base exudes serum and pus. The pure pus is a dull yellow, but contamination with blood may give it a salmon pink appearance.

Dangerfield and Gear [1] stated that in almost all their cases "the secondary lesions started within one or two weeks, along the course of the lymphatics." The earliest nodes become larger and softer. If treatment has not been instituted, the overlying skin tends to become adherent and then to break down. This results in the formation of a granulomatous ulcer akin to the primary lesions.

PATHOLOGIC CHARACTERISTICS

The histologic picture is not diagnostic but is typical only of a granuloma. In Bedell's case the report of biopsy stated that "subacute and chronic inflammation" was present and that "lymphoma cannot be excluded." Du Toit stated that the essential pathologic feature is a reticuloendothelial response by the tissues, with the proliferation of these cells and the formation of nodules. Scattered among the reticuloendothelial cells are plasma cells, lymphocytes, occasional eosinophils and some multinucleated giant cells of the foreign body type. Characteristic follicles tend to be scattered throughout the lesion. These have a peripheral zone or reticuloendothelial cells with, in most cases, a central zone of neutrophilic polymorphonuclear cells. At the periphery of the nodule multinucleated giant cells are seen.

In Hill's case,[47] Verhoeff's report on the biopsy reads:

> . . . [the] tissue contains large number of foreign body giant cells, but no tubercles resembling T. B. or syphilis. Some of the tissue shows considerable infiltration with lymphocytes and some only moderate infiltration with these cells. By ordinary methods of staining no organisms could be found in the tissues; but Verhoeff's modified gram stain shows filamentous organisms scattered through the tissues, some of great length. Some of these are surrounded by giant cells, others are not. The lymph gland shows the lymph sinuses distended with epithelioid cells.

IMMUNITY AND IMMUNE REACTIONS

Sporotrichosis has been reported among persons of practically all races. However, as I have previously stated, it is not very common in the tropics; nor has it been considered common among Negroes in Africa.

Moore and Manting[57] cited Fox and Lain, who in their wide experience among American Indians have never seen a case. Moore and Manting reported a case in a half-breed (French-Indian).

A positive reaction to the agglutination test may be demonstrated, but the value of this test has been nullified by the fact that the spores have been similarly agglutinated by the serum of normal controls. Bloch, cited by Lewis and Hopper,[58] expressed the belief that the intracutaneous test with an extract of Sporotrichum is valuable. This gave a positive reaction in our case. De Beurmann stated that a negative reaction rules out the diagnosis but that an occasional false positive reaction may occur. Du Toit stated the opinion that a positive cutaneous reaction is characteristically found in cases of the disease. In the experimental production of the disease in a volunteer, a positive cutaneous reaction occurred on the fifth day following inoculation.

Du Toit stated that the serum from patients with the disease agglutinated a suspension of the spores to a high titer (1:600) but that

the serum of some "normal" patients did almost the same. Therefore he felt that the complement fixation test was unreliable.

This same author found that the white rat was the most susceptible laboratory animal. When the males were given intraperitoneal inoculations, a typical scrotal reaction developed within ten days. The scrotum became swollen and inflamed and the testicles enlarged and fixed. Abscesses occurred in the peritoneum and the scrotum. When the animals were inoculated in the tail tip, the abscesses could be seen spreading up the tail.

DIFFERENTIAL DIAGNOSIS

Prior to the development of the chain of lesions there is nothing characteristic of the chancre which suggests sporotrichosis. When the chain of nodules or ulcers makes its appearance, sporotrichosis is suspected, although the other granulomas must be ruled out.

Syphilis and tuberculosis can be ruled out on the basis of the laboratory tests, the clinical pictures and the specific lesions seen at biopsy. Leprosy falls into somewhat the same category. When the conjunctiva is involved, it is often difficult to rule out Parinaud's conjunctivitis. Verhoeff reported the presence of Leptothrix in cases of this disease, and this organism has characteristics which differ from those of the sporotrichum. In cases of the oculoglandular type of tularemia the diagnosis may be confirmed by isolation of the organism from inoculated guinea pigs or by isolation of the specific agglutinins for Pasteurella tularensis in the patient's blood. When the conjunctiva is involved, trachoma must also be ruled out. Here, the typical conjunctival and corneal lesions, as well as the results of laboratory studies, are of aid.

The positive diagnosis of sporotrichosis rests on the identification of the organism. Direct smears of material taken by aspiration, preferably of an unopen nodule, may show the typical cigar-shaped organism as it appears in the tissues. However, most observers agree that direct smears are unsuccessful in too high a percentage of cases. Aspiration of the unopen nodule, followed by culture on Sabouraud's medium (or one of its modifications), will usually reveal the organism when examination is made with a hanging drop. The pus may be streaked on agar, but in our case this did not show any growth of the organism. On Sabouraud's medium the growth is usually recognizable in from three to five days. In early stages the colonies are small and white, with no aerial myceliums. As the growth increases, the surface of the colony becomes moist, wrinkled and membranous. The color may vary from cream to black.

Du Toit [2b] stated that he kept the cultures at room temperature. The early colonies are white and star shaped.

These enlarge in a few days forming a colony of about one-half inch in diameter. The center of the colony is thrown into irregular folds, while smooth regular folds radiate down from the raised center to the smooth, flat periphery. After a few days a brownish-black pigment appears in the center of the colony. The appearance of these colonies is so characteristic as to allow diagnosis to the naked eye.

George Lewis has confirmed this observation.

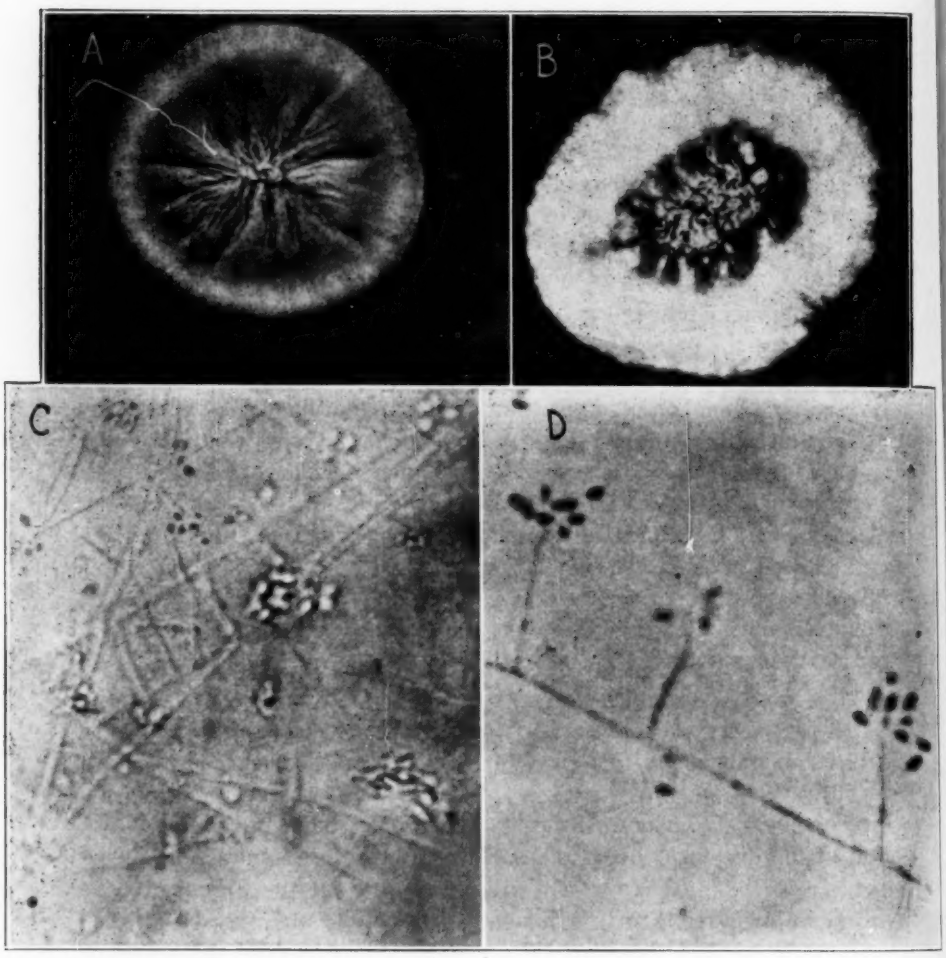

Fig. 3.—Two strains of Sporotrichum schencki, showing a variation in the appearance of the colonies. *A*, stock strain after two weeks. *B*, primary culture after four weeks; *C*, culture mount showing clusters of spores and filaments (× 390); *D*, culture mount showing clusters of spores borne at the tips of short stalks (× 715). From Lewis and Hopper,[58] page 305.

Microscopically, the spores are seen to germinate in about twenty-four hours at one or both poles, sending out long filaments, which in a few days show much interlacing branching. The fungus appears as

delicate (2 microns in width), branching, septate hyphae, which bear conidia laterally or in groups on the ends of the lateral branches. The conidia appear in clusters of from 2 to 20 or as single spores. They are ovoid to spherical. In old cultures they are altered and appear as round, thick-walled structures, measuring from 2 by 4 to 2 by 6 microns.

If culture does not yield the organism, inoculation of laboratory animals may help, as I have pointed out earlier in this paper. Guinea pigs are usually immune, and white rats are best for inoculation.

PROGNOSIS

Most patients with sporotrichosis will respond well to treatment, even when seen with the disease in a fairly advanced state, provided that the deeper structures of the body are not yet involved. Implication of the globe (unless by direct perforation by the traumatizing and etiologic agent) is evidence of deep invasion and indicates that the organism has reached the blood stream. Numerous fatalities have been reported.

TREATMENT

Administration of potassium iodide is the treatment of choice. In all the reported American cases of ocular sporotrichosis the disease responded to this drug, with the exception of the cases of Hill and Sanford Gifford. Gifford advised administration of 4 drachms (15.5 Gm.) daily. The drug is usually given in the form of the saturated solution, the initial dose being 10 drops three to four times a day, and this amount is stepped up 5 drops per dose per day until the limit of tolerance has been reached. The treatment should then be continued at this level for several weeks and should not be stopped immediately with the appearance of a clinical cure. Most observers agree that the average period of medication is from one month to about six weeks. The iodides have no direct action on the sporotrichum. Davis expressed the belief that fibroblastic elements are stimulated and that through their proliferation and encapsulation a cure results. Iodides have been frequently used locally and by direct injection, in addition to their use orally. Roentgen therapy has also been used, but there is no evidence that it is of any great value in this disease.

Lewis [58] believed that surgical procedures are contraindicated, as they are of no benefit, except possibly for a single incision in the presence of fluctuation. He also suggested that wet dressings of solution of aluminum acetate N. F. (1:15) or of potassium permanganate (1:3,000) may be applied for a half-hour several times daily. Gentian violet medicinal in a 1 per cent aqueous solution may be used to irrigate the lesions, or strong solution of iodine U. S. P. in half strength

concentration may be applied. Lewis also suggested that thymol be tried with those patients who cannot tolerate iodides.

Noojin and Callaway [59] recently reported on the effectiveness of the sulfonamide compounds in vitro on Sporotrichum schenckii. They found sulfanilamide the most effective of the nine sulfonamide preparations which they used. They stated that it almost completely inhibited the growth of the fungus in vitro. It would seem, however, that the iodides should be given a fair trial in the treatment of sporotrichosis before other forms of therapy are resorted to.

SUMMARY

Forty-eight cases of ocular sporotrichosis have been reported in the world literature, including the case reported in this paper. Apparently in 34 of these cases the ocular disease was primary and in 15 secondary to involvement elsewhere in the body. In 2 of these cases (Nino and Sobly Bey [54]) the diagnosis was in question.

The cases are distributed anatomically as follows: lids, 17; conjunctiva, 10; lacrimal sac, 2; conjunctiva and lacrimal apparatus, 1; brow, 1; intraocular, 5; orbit and lids, 5; cornea, 3; limbus, 1; uveal tract (without other lesions), 1, and lacrimal canal, 1.

Of the 48 reported cases, 10 have occurred in the United States and the majority of the remainder in France.

There are several excellent reviews of ocular sporotrichosis in the world literature. The first was that of Toulant,[60] who listed all the 23 cases occurring prior to 1913. Wilder and McCullough gave the first complete review in the American literature. This was followed almost immediately by Bedell's fine presentation. In 1922 Sanford Gifford again brought the literature up to date, with his first paper on the subject. Hill performed the same service in 1928. This is the last complete review of ocular sporotrichosis in the literature. It has been my attempt in this paper to bring the subject up to date again.

525 East Sixty-Eighth Street.

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59. Noojin, R. O., and Callaway, J. L.: Effectiveness in Vitro of Sulfonamide Compounds on Sporotrichum Schencki, Arch. Dermat. & Syph. **49**:305 (May) 1944.

60. Toulant, P.: Essai sur la sporotrichose oculaire, Thesis, Paris, 1912-1913, p. 390.

A POSITIVE CONTACT BALL AND RING IMPLANT FOR USE AFTER ENUCLEATION

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THIS IS a preliminary report on a new type of implant based on one year's use. This implant has a positive mechanical contact between the implant and the prosthesis, which provides a wide range and spontaneity of movement. The implant, in addition, eliminates sagging of the lower lid and minimizes sinking in of the upper lid.

Previously described implants, with the exception of the combined implant and prosthesis recently described by Ruedemann,[1] have been totally enclosed within Tenon's capsule and the conjunctiva or, in the case of evisceration, within the sclera and conjunctiva. In these circumstances, the available conjunctiva left to permit the prosthesis to rotate in the fornices has been diminished from that in the natural eye by the area occupied by the cornea. In the implant to be described, the anterior face is exposed, and thus the fornices have not been diminished. The exposed face, with an opening to receive a pin on the prosthesis, also permits mechanical contact and transmission of movement to the prosthesis.

Since the prosthesis does not rest on the lower lid at all, but is supported through its mechanical fitting to the implant, there is no sagging of the lower lid. Although it is not possible to be certain at the present time that sinking of the upper lid will not take place with this type of implant, it has not been sufficient in the patients so far operated on to require correction.

DESCRIPTION OF IMPLANT AND PROSTHESIS

Implant.—The original design of the implant (fig. 1) contemplated a plastic (methyl methacrylate) sphere 20 mm. in diameter, narrowed anteriorly to permit the attachment of a metal ring approximately 19 mm. in diameter, to which the four rectus muscles are attached. The ring is made of this size because this is the approximate diameter of the eye at the insertion of the rectus muscles. The anterior face of the implant is flattened and has a depression into which a gold pin is snugly, but not tightly, fitted. Originally these implants were made of three parts, put together by two screws at the operating table. However, it was not found practicable to do this, and Mr. Cheetham, the dental technician at the

1. Ruedemann, A. D.: Read at the Eighty-First Annual Meeting of the American Ophthalmological Society, Nov. 12, 1945.

Army hospital where this work was done, was able to make the implant in one piece. These implants required custom fitting of the pin when they were individually made. It is planned to make the entire implant of vitallium, so that the parts are interchangeable. The size of the ball is made 20 mm. to allow the prosthesis to overlap when fitted and still not to be larger than the normal eye. The anteroposterior diameter is made 15 to 16 mm. to allow space in front of the implant for the artificial eye. Later models of the implant have holes drilled in the ring to

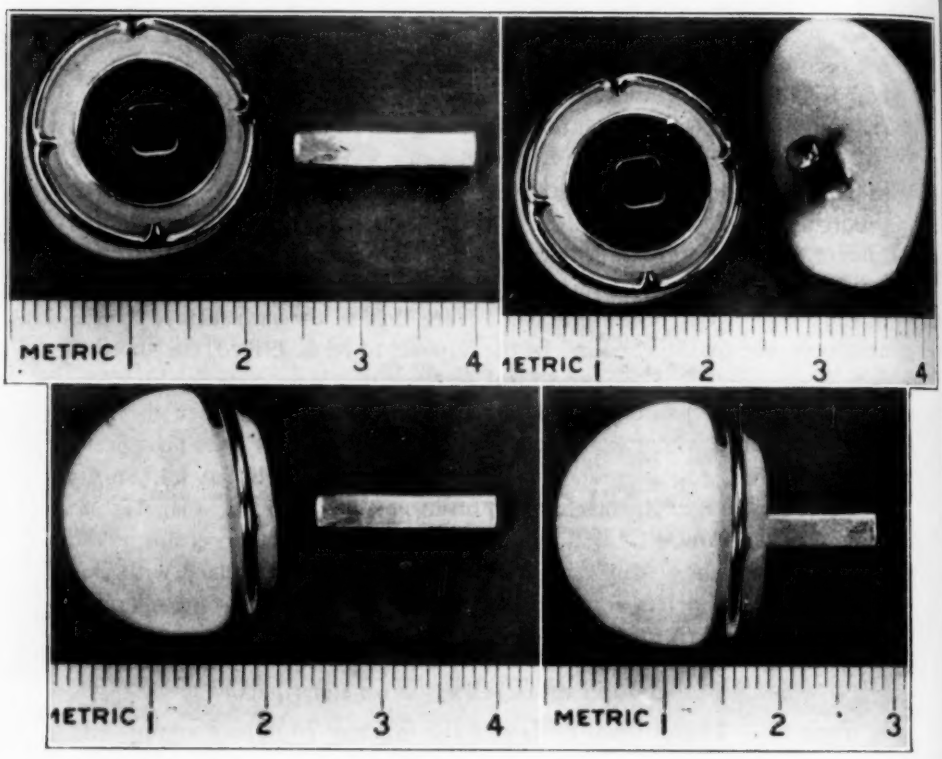

Fig. 1.—*A*, face view of implant, showing opening to receive the pin which is later attached to the prosthesis and the ring to which the rectus muscles are sutured; *B*, face view of implant and posterior view of prosthesis with pin attached; *C*, side view of implant and pin; *D*, side view of implant with pin in place.

allow tissue to grow through and also slight projections on the inner aspect to hold the muscles in place during operation.

Small implants, 14 mm. in diameter, with a 14 mm. ring, were made to place in sockets from which a sphere had been removed. In this case the muscles were not isolated. It was found that the movement of the stump was totally transmitted to the prosthesis, but no more movement was developed than was present in the stump originally.

Prosthesis.—The pin which is made with the implant is attached to a plastic artificial eye. It is necessary to center the iris carefully at the time of manufacture of the eye. The diameter of the eye has to conform as nearly as possible to the diameter of the other eye, otherwise the rotation of the eye would be unnatural in extreme positions, leaving a space either between the eye and the lids or between the eye and the socket. This is a decided departure from the normal procedure in making artificial eyes, in which the diameter is often made larger than the normal in order to obtain good contour of the lids. The eye is trimmed down until it is just large enough to stay inside the lids and show no edge on extreme movement to the right or left.

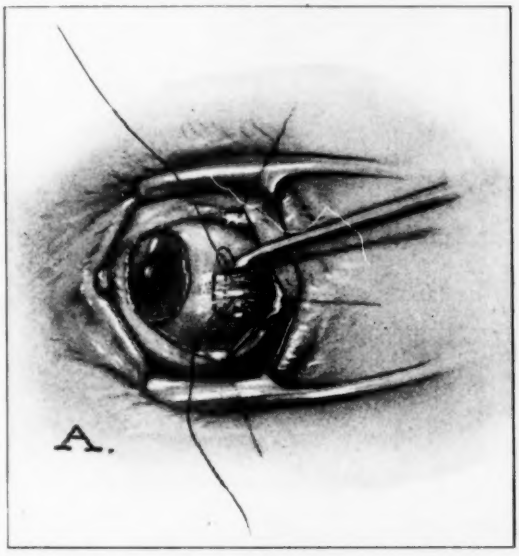

Fig. 2.—Lateral rectus muscle isolated and 00 silk suture passed through tendon. A whip stitch is not necessary.

OPERATION

As with any enucleation, the procedure may be done with local anesthesia, using 2 per cent procaine hydrochloride and epinephrine for a retrobulbar injection, or with the patient under general anesthesia. In the latter case a retrobulbar injection reduces the amount of anesthetic used.

After routine preparation of the field, a Weeks speculum is inserted, and the conjunctiva is incised at the limbus with Stevens scissors and the dissection carried well back to the fornices. One of the rectus muscles is then picked up with a muscle hook and isolated, as in a muscle operation. A single-armed 00 black silk suture is passed through the

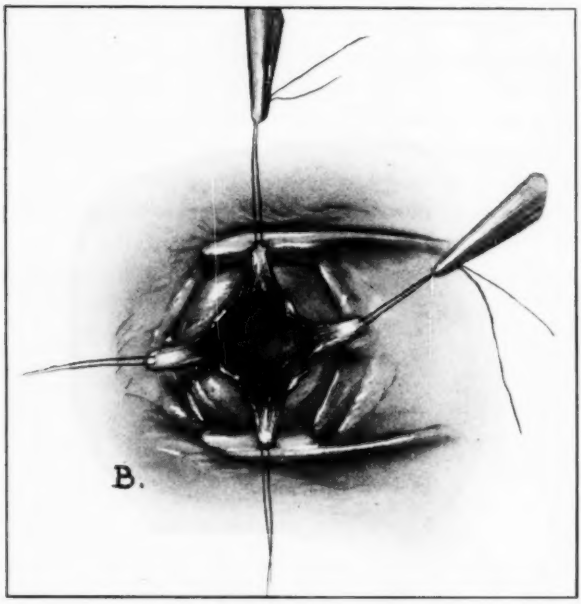

Fig. 3.—Enucleation complete.

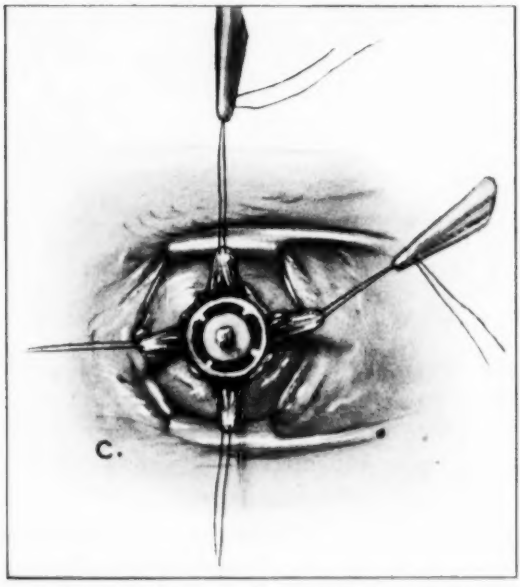

Fig. 4.—Ball and ring implant placed in Tenon's space.

tendon and the muscle cut from its insertion (fig. 2). This procedure is then repeated for the other three rectus muscles (fig. 3). The enucleation is then completed in the usual manner and the eye delivered.

Tenon's capsule is inspected by holding the margins forward with fine-toothed forceps, and the implant is then put into Tenon's space (fig. 4). The assistant now holds the implant in place by grasping the pin with Kelly forceps. The four silk muscle sutures are now passed through

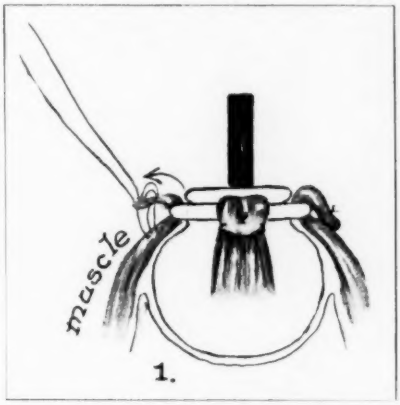

Fig. 5.—Drawing showing the manner in which the rectus muscles are carried through the ring and held in place with mattress sutures.

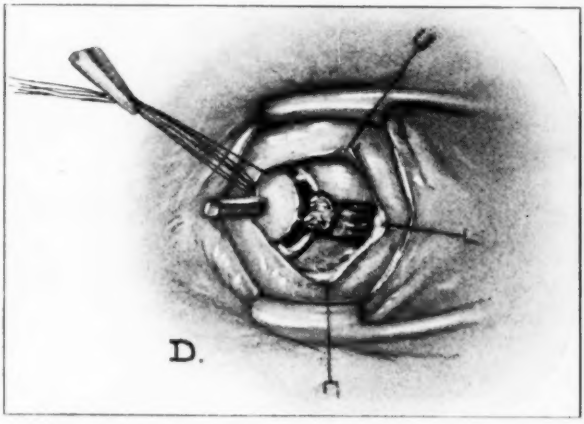

Fig. 6.—A rectus muscle sutured in place in the ring.

the ring from below upward in their proper place. The tendon of the inferior rectus muscle is grasped with von Graefe fixation forceps, and traction is made until approximately 4 mm. of the muscle is through the ring. Both needles, (½ inch [12 mm.] curved) of a double-armed 000 black nylon mattress suture are passed through the muscle just below the ring, from below upward. Both needles are then passed

through the tendon (fig. 5). The fixation forceps are then given to the assistant, who holds the muscle downward so that the muscle is looped around the ring. The suture is now triple-tied and cut (fig. 6). The fixation forceps and the original black silk suture are removed. This procedure results in the muscle being looped around the ring from below upward and tied on itself. The three other rectus muscles are sutured in a similar manner (fig. 7).

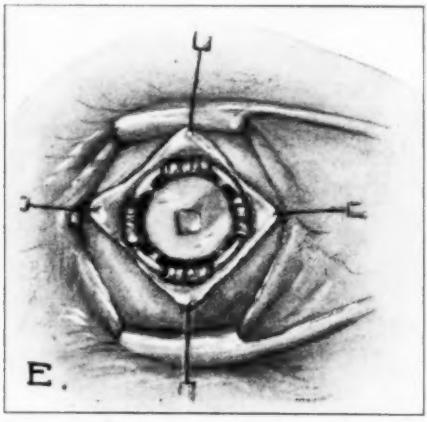

Fig. 7.—The four rectus muscles in place.

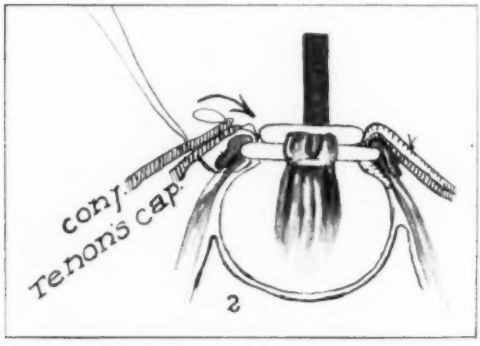

Fig. 8.—Drawing showing how the conjunctiva and Tenon's capsule are sutured around the ring, the sutures being passed in the reverse direction to those for the rectus muscles.

Five to eight double-armed 000000 black silk sutures are now passed through the conjunctival edge from without inward and in the same direction through a firm part of the anterior edge of Tenon's capsule. Both needles are then carried under the ring and out through Tenon's capsule and the conjunctiva (fig. 8). When these sutures are pulled and tied, Tenon's capsule and the conjunctiva are rolled inward around

the ring on themselves. The central part of the implant remains exposed (fig. 9).

The pin is now removed, and a small amout of petrolatum gauze is placed inside the lids and an adhesive dressing (Wheeler patch) applied.

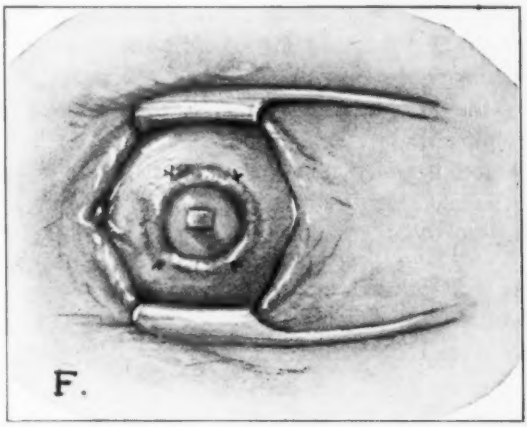

Fig. 9.—Completed operation. The anterior face of the implant is left exposed.

Fig. 10.—Photographs showing the extremes of movement, with prosthesis in the right socket.

POSTOPERATIVE CARE

The patient is allowed to be elevated or turned as desired and is allowed up as soon as he feels like it, usually in one or two days. The dressing is changed on the fifth and eighth days. No dressing is used after the eighth day. The conjunctiva–Tenon's capsule sutures are removed about the twelfth day, or whenever convenient. The patient is ready for fitting of the prosthesis in eighteen to twenty-one days.

COMPLICATIONS

In 3 cases implants were made in three parts—in all these the implant had to be removed. In 1 of the cases the patient had mild pneumonia and orbital cellulitis six months after operation, necessitating removal of the implant. No organisms were grown from his conjunctival sac. Two other implants had to be removed because the ring became exposed, owing, it was thought, to lack of firm suturing at the time of operation. Surgical gut was not found satisfactory for suturing the muscles. Incidentally, eight different ophthalmologists performed the operation during the developmental period.

It was not found practicable to resuture Tenon's capsule if the ring became exposed. Neither was it possible to use the implant in a case of scarring or other complications—the sutures would not hold.

Fig. 11.—Photographs similar to those in figure 10, with prosthesis in the right socket.

If a muscle became exposed, it inevitably let go, allowing the implant to prolapse. Firm and properly placed sutures are very important. One smaller implant was placed in the sclera after evisceration but did not seem to give any better, if as good, results.

RESULTS

Twenty-two patients were operated on. Five implants were removed, for the reasons already stated. The amount of movement is indicated in the accompanying tabulation (figs. 10 and 11).

	Horizontal Movements	
Maximum	Minimum	Average
75°	65°	70°
	Vertical Movements	
Maximum	Minimum	Average
65°	65°	65°

SUMMARY

A new implant embodying a new direct method of transmission of movement is described. This implant gives to the prosthesis a larger range and greater spontaneity of movement than any previously used. The operative procedure, postoperative care and complications are discussed.

There are only two places in the body where a similar inert material projects to the exterior—the teeth and the nails. Time will tell whether these implants will be tolerated. This preliminary report is made one year from the date of the earliest retained implantation (August 1945).

Miss Helene Cleare made the illustrations, and Mr. A. Cheetham assisted in manufacture of the implants. Dr. Stanley Erpf and Dr. R. D. Stephens assisted in adapting the plastic prosthesis to this type of implant.

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Clinical Notes

AN AID IN FACILITATING POSTOPERATIVE DRESSING OF THE EYE IN PATIENTS WITH AKINESIS OF LIDS

WILLIAM D. GILL, M.D., SAN ANTONIO, TEXAS

SEVERAL years ago I called attention to a simple method of maintaining postoperative closure of the eyelids after akinesis of the orbicularis oculi muscle had been employed, which consisted in sealing the lashes of the upper lid to the lower lid by means of a small droplet of collodion.[1] The method has worked satisfactorily over a period of years and is used routinely in my practice in all cases in which akinesis of the lids has been used. The possibility of an accident such as the patient's opening the eyelids beneath the dressing, with possible contact with the cornea, is prevented. With the collodion seal, the lids are maintained in approximation during the period that akinesis is in effect. The advantage over suture of the lids is evident. No additional wound is created; and the greater ease of use and the ease of removal, with absence of pain, will recommend its use. There is sufficient space on each side of the point of fixation of the eyelids with the collodion droplet to allow the introduction of medicaments, such as ointments, into the cul-de-sac and permit the escape of secretions from the eye. The chief objection to the use of this method has been the difficulty in opening the eye when the method had fulfilled its usefulness. At the time of the first postoperative dressing, when one desires to inspect the eye, it was formerly my custom to loosen the collodion fixation with a pair of finely pointed tissue forceps. This method is satisfactory but has certain objectionable features. More recently, in order to facilitate opening the lids and breaking the collodion seal, a short length of silk suture, 1½ to 3 inches (3.8 to 7.6 cm.) in length, is introduced beneath the lashes after the eyelids have been closed and sealed with collodion. This can be accomplished before the sealing, but in such instances particular care has to be exercised to avoid incorporating the silk suture in the collodion droplet. When this silk suture is in position, its ends are brought together and draped onto the cheek below the eyelid; the two ends are tied together, and the customary dressing is applied. When the time comes to inspect the eye, it is a simple matter to grasp the ends of the silk suture and, with gentle traction, to separate the lids, as the suture will find its way between the lashes and the lower lid and the collodion seal will be easily and painlessly overcome. The small fragment of collodion usually remains adherent to a few of the lashes of the upper eyelid and can be pulled away with a small dressing forceps. The additional feature of using the silk thread to break the collodion seal has been most satisfactory; it has greatly facilitated the

1. Gill, W. D.: A Way to Keep the Eyelids Closed After Infiltration Anaesthesia, Ann. Ophth. **58**:269, 1928.

postoperative inspection of the eye and is considered less barbaric than the commonly employed lid suture and is worthy of more general use.

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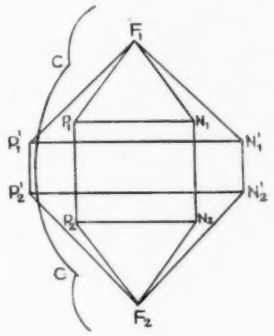
CARDINAL POINTS IN APHAKIA

JOSEPH I. PASCAL, M.D., NEW YORK

IN TWO previous communications [1] it was shown how the location and displacement of the cardinal points of the eye can be visualized and remembered by the use of the benzene ring. It was shown that when the eye accommodates the new position of the cardinal points can be represented by a long, slender ring situated inside the regular ring. When the crystalline lens is removed from the eye, the optical system of the eye is radically altered. Its refractive power has been decreased by about one fourth, and there has also been a shifting of the cardinal points. It can be shown similarly that the location and displacement of



Cardinal points in the normal and the aphakic eye.

the cardinal points in the aphakic eye can be represented by a broad and flattened ring drawn from the regular ring representing the normal static eye.

In the accompanying figure, the principal and nodal points of the aphakic eye are indicated by adding a prime to the corresponding letters P and N. It is seen that the focal lengths of the aphakic eye are longer, as shown by $P'_1 F_1$ and $P'_2 F_2$. This would be expected, since the eye is now a weaker refractive system. The two principal points P'_1 and P'_2 have moved forward from P_1 and P_2 toward the cornea and are closer together. In fact, they have moved forward so as to be a little in front of the cornea, as shown in the diagram. (They are also crossed; i. e., P'_2 is in front of P'_1—an interesting detail not shown.) The nodal points N'_1 and N'_2 have moved backward from N_1 and N_2, away from

1. Pascal, J. I.: Cardinal Points in the Static and in the Dynamic Eye, Arch. Ophth. **34**:319 (Oct.) 1945; A Memory Scheme for the Cardinal Points, ibid. **22**:448 (Sept.) 1939.

the cornea, and are also nearer together. This produces less displacement of the axial rays.

The greater separation of $P'N'$ shows the longer effective "radius," which would replace the whole optical system of the aphakic eye. The backward movement of the rodal points shows that in the aphakic eye the retinal image is smaller than in the phakic eye. Of course, the retinal image is blurred; but disregarding the enlargement due to the diffusion circles and taking the image size only from the centers of the diffusion circles, one sees that the retinal image is smaller in the aphakic eye, since the nodal points are closer to the retina. The optical image, of course, is greatly enlarged.

37 West Ninety-Seventh Street.

Obituaries

THEODORE LASATER TERRY, M.D.
1899-1946

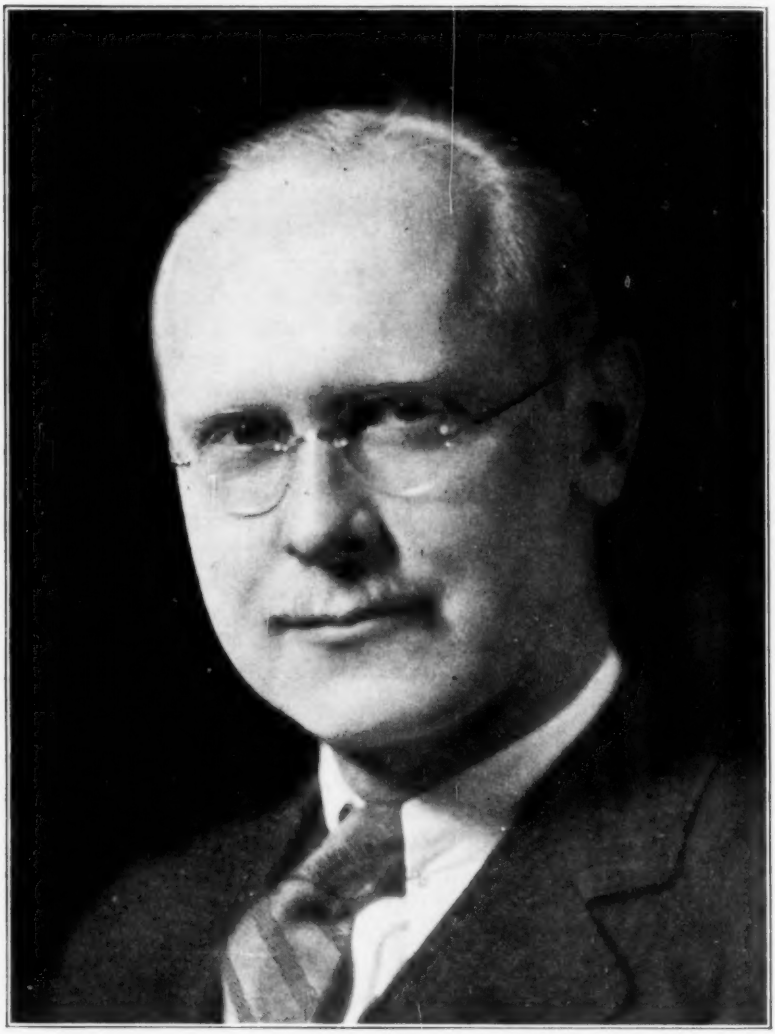
Theodore Lasater Terry died suddenly of cardiac failure in Boston on Sept. 28, 1946, in his forty-seventh year. Thus ended, all too early, a brilliant career in ophthalmology. Dr. Terry was born in Ennis, Texas, Feb. 19, 1899, the son of John S. and Lucy Lasater Terry, the former a respected physician in his community.

He received his early schooling in Ennis, attended the Southern Methodist University and studied medicine at the University of Texas, from which he received his degree of Doctor of Medicine in 1922. After interning at the Henry Ford Hospital, in Detroit, he returned to the University of Texas to study general pathology, eventually becoming an associate professor in 1926, It was during this period that his interest in ophthalmology began, as indicated by the title of his first published paper in the *Texas State Medical Journal,* "Neurogenic Tumors of the Retina and the Optic Nerve." He completed the residency in ophthalmology at the Massachusetts Eye and Ear Infirmary in 1928 and was appointed assistant pathologist at that institution. Here he studied under Dr. Frederick H. Verhoeff, eventually becoming pathologist when Dr. Verhoeff assumed the directorship of the Lucien Howe Laboratory of Ophthalmology in 1932. He also rose through the clinical grades at the Infirmary, becoming clinical assistant in 1929, assistant surgeon in 1932 and surgeon in 1939.

Dr. Terry's accomplishments were so varied that in describing them it is difficult to know where to begin. He was an excellent clinician, surgeon and pathologist, but perhaps his greatest forte was teaching. Here he was completely at home, imparting to his students his own enthusiasm for the subject. He had strong convictions about the obligations of well trained ophthalmologists to give aid and counsel to the younger men. He was not one to be satisfied with a system of teaching just because it had always been in vogue, but was constantly trying new methods, most of which were thoroughly sound. At the time of his death he was assistant professor of ophthalmology at the Harvard Medical School, in charge of postgraduate teaching, and during the war had acted as head of the department, which he administered in excellent fashion in spite of many difficulties.

In the field of clinical research his name was well known, his chief studies dealing with keratoconus, malignant melanoma and ocular mal-

development in premature infants. Retrolental fibroplasia received the major part of his attention during the past few years. He devoted considerable time to clarifying this syndrome and bringing it to the attention of members of the medical profession as one of the leading causes



THEODORE LASATER TERRY, M.D.

1899–1946

of infant blindness in the country. In 1942 he received the certificate of merit from the American Academy of Ophthalmology and Otolaryngology for clinical research in this field. Just before his death, he had

planned an extensive research program in an effort to ascertain the cause of this malady among infants.

Dr. Terry was a faithful attendant at medical meetings, taking an active part in discussing papers of others, as well as contributing many of his own. He was the author of many scientific papers, covering a wide variety of subjects. A complete list is appended.

He was a member of the American Board of Ophthalmology; the American Ophthalmological Society; the American Academy of Ophthalmology and Otolaryngology; the American Medical Association, Section on Ophthalmology; the American College of Surgeons, the American Association for the Advancement of Science and the New England Ophthalmological Society, serving as past president of the last organization. He was also a trustee of the Association for Research of Ophthalmology, Inc., and president of the Foundation for Vision, which was formed this year to continue research on retrolental fibroplasia and other ophthalmologic problems.

There was another side to Dr. Terry besides the scientific. His home and family meant everything to him. He was interested in photography, travel, current events, economics and politics, and in spite of the terrific demands his profession made on his time he did not fail to keep well informed about the outside world. He also felt strongly about doing his part in national defense, and not many of his colleagues were aware that he held a commission as lieutenant in the Medical Corps of the United States Naval Reserve until ill health forced his resignation, several years before the war. He had a keen sense of humor and was a delightful companion.

To his family and friends, to the institution he served and to ophthalmology in general, his loss is a tremendous blow.

EDWIN B. DUNPHY.

BIBLIOGRAPHY

Terry, Theodore L.: Neurogenic Tumors of the Retina and Optic Nerve: Gliomata, Texas State J. Med. **19**:379-382 (Nov.) 1923.

X-Rays Ineffective Against Herpes Virus, Am. J. Ophth. **2**:544 (July) 1928.

The Contact Glass: Its Use in Conical Cornea, and Errors of Refraction, Guildcraft **5**:2, 1931.

Histologic Changes in an Eye Eight Years After Sclerocautery Puncture of the Retina, Arch. Ophth. **8**:182-185 (Aug.) 1932.

——and Mysel, P.: Sarcoma of Eyelid: Metaplasia of Leiomyosarcoma to Round Cell Sarcoma After Repeated Attempted Excisions, ibid. **12**:689-692 (Nov.) 1934.

——and Dunphy, E. B.: Metastatic Carcinoma in Both Optic Nerves Simulating Retrobulbar Neuritis, ibid. **10**:611-614 (Nov.) 1933.

——and Mysel, P.: Pulsating Exophthalmos Due to Internal Carotid–Jugular Aneurysms: Use of Thorium Dioxide Sol in Localization, J. A. M. A. **103**: 1036-1041 (Oct. 6) 1934.

Angioid Streaks and Osteitis Deformans, Tr. Am. Ophth. Soc. **32**:555-573, 1934.

——and Johns, J.: Uveal Sarcoma—Malignant Melanoma: Statistical Study of Ninety-Four Cases, Am. J. Ophth. **18**:903-913 (Oct.) 1935.

Diathermy in Cataract Extraction, ibid. **19**:1105 (Dec.) 1936.

——and Fred, G. B.: Abnormal Arteriovenous Communication in the Orbit Involving the Angular Vein, Arch. Ophth. **19**:90-94 (Jan.) 1938.

A Modified Corneal Clamp to Facilitate the Insertion of Stitches, ibid. **20**:91-94 (July) 1938.

Some Physiological and Anatomical Aspects of the Cornea Affecting Its Pathology, Am. J. Ophth. **22**:153-155 (Feb.) 1939.

——Chisholm, J. F., Jr., and Schonberg, A. L.: Studies on Surface-Epithelium Invasion of the Anterior Segment of the Eye, ibid. **22**:1083-1110 (Oct.) 1939.

Care of the Eyes, in Health at Fifty, edited by W. Robey, Cambridge, Mass., Harvard University Press, 1939.

Malignant Melanoma—So-Called Sarcoma of Uvea: II. Problems in Diagnosis, Arch. Ophth. **22**:989-1022 (Dec.) 1939.

——and Mattis, R. D.: A Plastic Transillumination, ibid. **23**:164-165 (Jan.) 1940.

Malignant Melanoma: So-Called Sarcoma of Uvea: III. Extension into the Optic Nerve, ibid. **24**:206-214 (July) 1940.

——and Chisholm, J. F., Jr.: Studies on Keratoconus Relative to the Effect of Prolonged Application of Pressure, Am. J. Ophth. **23**:1089-1112 (Oct.) 1940.

Common Glaucoma Operations: Analysis Based on Histologic Findings, New York State J. Med. **41**:467-469 (March) 1941.

Truss for Application of Pressure to the Eye, Am. J. Ophth. **25**:333-334 (March) 1942.

Extreme Prematurity and Fibroblastic Overgrowth of Persistent Vascular Sheath Behind Each Crystalline Lens, ibid. **25**:203-204 (Feb.) 1942.

Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Premature Infants: II. Report of Cases—Clinical Aspects, Arch. Ophth. **29**:36-53 (Jan.) 1943.

Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Infants Born Prematurely: I. Studies in Development and Regression of Hyaloid Artery and Tunica Vasculosa Lentis, Am. J. Ophth. **25**:1409-1423 (Dec.) 1942.

Fibroblastic Overgrowth of Persistent Tunica Vasculosa Lentis in Premature Infants: IV. Etiologic Factors, Arch. Ophth. **29**:54-68 (Jan.) 1943.

Some Clinical Applications of Fluorescence in Relation to Melanotic Pigment, Am. J. Ophth. **26**:536-539 (May) 1943.

Retrolental Fibroplasia in Premature Infants: V. Further Studies on Fibroplastic Overgrowth of the Persistent Tunica Vasculosa Lentis, Arch. Ophth. **33**:203-208 (March) 1945.

Ocular Maldevelopment in Extremely Premature Infants: Retrolental Fibroplasia: VI. General Consideration, J. A. M. A. **128**:582-584 (June 23) 1945.

A Visual Defect of the Prematurely Born Infant, Outlook for Blind **39**:211-213 (Oct.) 1945.

Kinsey, V. E.; Jackson, B., and Terry, T. L.: Development of Secretory Function of Ciliary Body in the Rabbit Eye Evaluated from Ascorbic Acid Concentrations and Changes in Volume, Arch. Ophth. **34**:415-417 (Dec.) 1945.

Terry, T. L.: Further Consideration of Retrolental Fibroplasia, J. Pediat., to be published.

Correspondence

PERMEABILITY OF THE EXCISED CORNEA

To the Editor.—Researches reported by Drs. Holt and Cogan (The Cornea: VIII. Permeability of the Excised Cornea, as Determined by Measurements of Impedance, ARCH. OPHTH. **35**: 292 [March] 1946) have just come to our attention. The data in this paper, obtained through measurements of electrical impedance, definitely show that under the conditions used the corneal epithelium of excised beef corneas is virtually impermeable to various ions.

In an earlier paper in the same series (The Cornea: II. Transfer of Water and Sodium Chloride by Hydrostatic Pressure Through the Excised Cornea, ARCH. OPHTH. **27**: 696 [April] 1942) Cogan and Kinsey reported the results of osmotic studies concerning the semipermeability of the epithelium in excised cat corneas. In the latter paper, it was indicated in the authors' interpretation of the data that a difference in concentration of sodium chloride of approximately 0.03 per cent would be required to effect a transfer of fluid through the epithelium against a hydrostatic pressure of 400 mm. of mercury, whereas the theoretic osmotic equivalent of this hydrostatic pressure was stated to be 0.007 per cent sodium chloride solution. If, as indicated, a difference in sodium chloride concentration of more than four times the theoretic would be required to effect a transfer of fluid under these conditions, we should interpret this to mean that the epithelium was possibly somewhat sievelike with respect to sodium and chloride ions and that a high degree of semipermeability was not indicated.

Actually, the theoretic osmotic equivalent of 400 mm. of mercury is 0.07 per cent sodium chloride solution, not 0.007 per cent, as stated on page 703 of this paper. A reinterpretation of the data of Cogan and Kinsey based on this correction demonstrates almost as forcefully as the electrical impedance measurements in the recent study that the epithelium is virtually impermeable to sodium and chloride ions.

It is our interpretation that the curve in figure 5 of the paper by Cogan and Kinsey should be redrawn so that it passes through the zero line at a concentration difference of 0.07 per cent or greater, since it would be impossible to have an osmotic transfer of fluid below this concentration against a hydrostatic pressure of 400 mm. of mercury. If the curve is to meet this requirement and at the same time fit the experimental data, it cannot be drawn as a straight line. (There is, indeed, no necessity for drawing the curve as a straight line, since the amount of water that passes through a biologic membrane is not necessarily directly proportional to differences in concentration, irrespective of whether it is perfectly semipermeable. Experimentally it is difficult to avoid the establishment of concentration gradients in this type of system.)

If the curve in the figure is redrawn so that it passes through the points for differences in sodium chloride concentration of 0.080, 0.150 and 0.250 per cent and at the same time conforms to the aforementioned considerations, it will be found that the curve intersects the zero line at approximately a 0.07 per cent difference in sodium chloride concentration, even when adequate allowance is made for the probable experimental errors.

The interpretation therefore indicates a virtually complete impermeability of the epithelium to these ions and more thoroughly justifies the conclusions of Cogan and Kinsey that "the corneal epithelium, for a biologic membrane, possesses a high degree of semipermeability with respect to sodium chloride solutions."

W. O. Lundberg, Ph.D., Austin, Minn.
Hormel Institute, University of Minnesota.

Albert L. Anderson, Minneapolis.
Precision Contacts.

News and Notes

EDITED BY DR. W. L. BENEDICT

GENERAL NEWS

Eye-Bank for Sight Restoration, Inc.—The board of directors of the Eye-Bank for Sight Restoration, Inc., announce that Dr. Herbert M. Katzin, of New York, has been put in charge of the laboratory for ophthalmic research and that three fellowships have been granted for the purpose of studies of corneal vascularization, corneal pathology, vitreous replacement and vitreous transplant.

Annual W. Thornwall Davis Postgraduate Course in Ophthalmology. — The seventh annual W. Thornwall Davis Postgraduate Course in Ophthalmology will be held from Jan. 27 to Feb. 8, 1947, at the George Washington University School of Medicine, 1335 H Street, N. W., Washington, D. C.

During the first week there will be practical courses in ocular surgery, pathology and orthoptics. During the second week lectures will be given by F. Heed Adler, M.D.; J. Mason Baird, M.D.; S. Judd Beach, M.D.; Hermann Burian, M.D.; Ramon Castroviejo, M.D.; C. Alvin Clapp, M.D.; F. Bruce Fralick, M.D.; Deane B. Judd, PH.D.; Peter C. Kronfeld, M.D.; Walter I. Lillie, M.D.; Philip I. Salvatori, M.D.; Harold G. Scheie, M.D.; Edmund B. Spaeth, M.D., and Frederick W. Stocker, M.D.

For further information, apply to Ernest A. W. Sheppard, M.D., professor of ophthalmology, executive officer, 1801 K Street, N. W., District 4024, Washington, D. C.

Establishment of the Francis I. Proctor Laboratory for Ophthalmologic Research.—A sum of over $500,000 from the estate of Dr. Proctor has been given to the University of California by Mrs. Proctor for the establishment of the Francis I. Proctor Laboratory for ophthalmologic research. To assure adequate permanent space for the laboratory in the new science building which is to be constructed in the near future, Mrs. Berthold Guggenheim has guaranteed a sum of $65,000 toward the construction of this necessary space. In the interim the laboratory is being housed in temporary quarters. Dr. Michael Hogan has been appointed director of the laboratory.

SOCIETY NEWS

The Contact Lens Society.—The Contact Lens Society, purely scientific in object and organization, has been formed for the study of contact lens work in all its aspects. Members may be medical or nonmedical. The first scientific meeting will be held on Jan. 20, 1947, at 5:30 p. m., at the headquarters of the British Optical Association, 65 Brook Street, London, W. 1.

The officers of the society are as follows: president, Prof. Ida Mann; vice presidents, Mr. F. A. Williamson-Noble and Mr. K. Clifford Hall; joint secretaries, Mr. A. G. Cross and Mr. G. H. Giles; treasurer, Mr. C. H. Keeler. Other members of the council are Messrs. J. H. Doggart,

G. B. Ebbage and F. A. Juler; Sir Stewart Duke-Elder, and Messrs. F. Dickinson, H. B. Marton, G. D. McKellen and T. Hamblin.

The secretaries of the society, Mr. A. G. Cross and Mr. G. H. Giles, will be glad to supply information and forms of application for membership on receipt of a request addressed to 65 Brook Street, London, W. 1.

Washington, D. C., Ophthalmological Society.—The Washington, D. C., Ophthalmological Society will hold a meeting on Monday, Feb. 3, 1947, at 8 p. m. in the auditorium of the Medical Society of the District of Columbia, 1718 M Street, N. Dr. Peter C. Kronfeld, Chicago, will speak on "Selected Cases of Retinal Detachment," and Dr. F. Bruce Fralick, Ann Arbor, Mich., on "The Problem of Thyrotropic Exophthalmos."

Ophthalmological Society of Egypt.—The annual meeting of the Ophthalmological Society of Egypt will take place at the Memorial Ophthalmic Laboratory, Giza, Egypt, on Friday and Saturday, March 14 and 15, 1947, at 9 a.m.

Appointment of Medical Director of National Society for Prevention of Blindness, Inc.—The National Society for the Prevention of Blindness, Inc., announces the appointment of Dr. Franklin M. Foote as medical director. Dr. Foote has been district health officer of the Kips Bay–Yorkville Health District of the Department of Health of New York City and, before that, chief of the Division of Local Health Administration, Connecticut State Department of Health. He is assistant professor of public health and preventive medicine at Cornell University Medical College.

Dr. Foote is a native of Great Barrington, Mass., and received his degree of Doctor of Medicine from Yale University. During World War II he served as major in the Medical Corps of the United States Army and was principally occupied in teaching preventive medicine at the Medical Field Service School, Carlisle Barracks, Pa., and at the Brook Army Medical Center, Fort Sam Houston, Texas.

Centennial Meeting of the New York Academy of Medicine, Section of Ophthalmology.—The centennial meeting of the New York Academy of Medicine, Section of Ophthalmology, will be held March 17, 1947; Dr. Brittain F. Payne, 17 East Seventy-Second Street, chairman.

The subscription dinner (President's Gallery), at 6:15 p. m., will be followed by a meeting of the executive session (Hosack Hall), at 8:15 p. m.

The papers of the evening are as follows: "Past and Future Progress in American Ophthalmology," Dr. Derrick Vail (by invitation), Chicago; "History of Ophthalmology in Canada," Dr. John A. MacMillan (by invitation), Montreal, Canada; "History of the American Board of Ophthalmology," Dr. S. Judd Beach (by invitation), Portland, Maine; "New York's Contribution in the Development of Ophthalmology in This Country," Dr. Bernard Samuels, New York.

Correspondence may be addressed to the secretary, Dr. Milton L. Berliner, 57 West Fifty-Seventh Street, New York.

Ophthalmological Society of the United Kingdom.—The annual congress of the Ophthalmological Society of the United Kingdom will

be held in Glasgow, Scotland, on March 27, 28 and 29, 1947. The president, Dr. A. J. Ballantyne, will give an address entitled "De Senectute."

The subject for discussion will be "Rhinology in Relation to Ophthalmology," which will be opened by Dr. John Marshall (ophthalmic surgeon), Mr. Gilbert H. Howells (otorhinolaryngologic surgeon), and Dr. R. McWhirter (radiologist).

On Friday afternoon there will be a clinical meeting at the Glasgow Eye Infirmary.

On Saturday afternoon visits have been arranged to the Corporation of Glasgow Art Galleries, Kelvingrove, under the guidance of the director, Dr. T. J. Honeyman, and to the Hunterian Museum of the University of Glasgow, by invitation of the University Court.

The annual dinner of the society will be held on Thursday, March 27.

Honorary Secretaries are J. H. Doggart, 49 Wimpole Street, London, W. (council business); E. F. King, 79 Harley Street, London, W. (congress business).

UNIVERSITY NEWS

Graduate Training in Ophthalmology at the University of Toronto.—The department of ophthalmology of the University of Toronto has undertaken postgraduate training during the past year. This has been aided by a generous annual grant from Mr. Percy Hermant in establishing fellowships in ophthalmology. The postgraduate training for the fellows is now three years, of which the first is in the basic sciences of ophthalmology and the final two years are in clinical internships in one of the Toronto hospitals. Dr. J. R. Gaby, of Toronto, and Dr. A. W. Mahood, of Vancouver, British Columbia, Canada, have been appointed fellows for the current year. In addition to the fellowships, there are a number of clinical internships in ophthalmology of two years' duration.

The graduate instruction in ophthalmology in the hospitals in Toronto has been coordinated under the direction of the university. Didactic lectures are presented weekly to the interns by the members of the teaching staff in ophthalmology from October until May. On Saturday mornings ward rounds are made at the Toronto General Hospital and are attended by the interns in ophthalmology from the various hospitals.

The interns are rotated through the department of ophthalmology of the Hospital for Sick Children in order that they may become familiar with the medical and surgical procedures of children's ophthalmology.

The senior interns are given instruction in the preparation of scientific papers and their presentation, and each presents a paper at the Toronto Academy of Medicine, Section of Ophthalmology.

There have been a number of changes in the teaching staff of the department of ophthalmology of the university. Dr. Walter W. Wright has retired as professor of ophthalmology in the university and head of the department in the Toronto General Hospital and has been appointed professor emeritus. He has been succeeded by Dr. A. J. Elliot. Dr. A. E. MacDonald and Dr. A. Lloyd Morgan have been made assistant professors of ophthalmology, and the teaching staff of the department in the university has been increased by the appointments of Dr. J. S. Crawford, Dr. C. E. McCulloch, Dr. J. C. Hill and Dr. M. E. Albertson.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Biochemistry

DISTRIBUTION OF VITAMIN C IN THE LENS. H. E. HENKES, Ophthalmologica **108:** 11, 1944.

Cylinders were cut from the lenses of a large number of species of animals, and the ascorbic acid content of the various layers was determined by the microtitration method of Glick and Biskind (*J. Biol. Chem.* **110:** 1, [June] 1935). The highest values were found in the subcapsular cortex, and the nucleus had the least. The decrease in ascorbic acid content from capsule to nucleus differed in different species of animals. The nucleus of cataractous lenses had a very low ascorbic acid content. Guinea pigs with scurvy lost ascorbic acid in the cortex first, but this was quickly regained with appropriate feeding.

F. H. ADLER.

Conjunctiva

HEMORRHAGE FROM THE CONJUNCTIVA: NOTES ON A CASE OF CAPILLARY ANGIOMA. J. P. WHITE, Brit. J. Ophth. **29:** 635 (Dec.) 1945.

A married woman aged 40, apparently healthy, had had bloody tears from the right eye on two occasions. The bleeding was arrested by tightly applying a handkerchief to her eyelids. The attacks were unrelated to menstruation.

On eversion of the upper lid, a small reddish growth, about 3 by 2 mm., was noted on the palpebral conjunctiva near the superior margin of the tarsus. The biomicroscope showed that it was highly vascular. The growth, together with a piece of the tarsus, was excised. In the tarsal region the epithelium was flat and thin. In the area of the fornix the conjunctiva was laid in many folds and the epithelium was full of goblet cells. One fold in particular contained many thin-walled vessels. The reticulum was filled with lymphocytes.

It would appear, therefore, that the capillary angioma was the source of the hemorrhage.

The article is illustrated. W. ZENTMAYER.

Congenital Anomalies

OPERATION FOR CONGENITAL COLOBOMA OF THE UPPER LID. R. GAUFFRE, Arch. d'opht. **5:** 342, 1945.

Congenital coloboma of the upper lid presents an immediate indication for operation, especially in order to protect the cornea. The author describes the procedure which he used in the case of a child aged 4 years and in a second case, that of a child of 6 months. A drawing illustrating the procedure accompanies the article. He produces

akinesia in order to prevent voluntary closure of the lids, and this akinesia lasts ten days. He also uses a serum, which helps to avoid edema. These two procedures, he thinks, are contributory to the good results obtained.

S. B. MARLOW.

Cornea and Sclera

SUPERFICAL PIGMENTATION OF THE CORNEA. R. R. BLONDIS, Am. J. Ophth. 29: 316 (March) 1946.

Blondis reports 8 cases of pigmentation of the cornea. He states that the condition is not rare and believes it to be due to melanin which invades the cornea from the conjunctiva by way of the lymph spaces. Conclusive evidence of the character of the pigment is not yet available.

W. S. REESE.

TRANSPLANTATION OF CORNEA. O. I. SHERSHEVSKAYA, Am. Rev. Soviet Med. 2: 525 (Aug.) 1945.

Shershevskaya states that of the 435 keratoplasties performed in Filatov's clinic between 1922 and 1938 success was obtained in 24.1 per cent with the use of corneas from living donors. Filatov and his school developed the use of preserved cadaver corneas. In the Odessa clinic 264 transplantations were done in which this method was used. The successful results in this series amounted to 67 per cent. Filatov perfected the technic of the operation, suggested a series of new methods, constructed instruments which made the operation simpler and easier and widely popularized keratoplasty. His results have aroused the attention of ophthalmologists. Corneal transplantation is being performed in many hospitals throughout the Soviet Union.

J. A. M. A. (W. ZENTMAYER.)

A RARE FORM OF KERATITIS OF PROBABLE STREPTOCOCCIC ORIGIN. P. MATA LOPEZ, Arch. Soc. oftal. hispano-am. 4: 252 (April) 1945.

In his many years of practice, the author has seen 3 cases which he has classified in the category designated by the title of this article. The condition is characterized by coordinated lesions of the skin and cornea in a fashion that he has never seen described before. The cutaneous lesion which initiated the condition in his 3 cases had been present for months before the onset. It is an eczematous dermatitis, proved to be of streptococcic origin, located in the retroauricular fold and giving slight symptoms. The corneal lesion then made its appearance, in the form of a superficial, infiltrated, ulcerative keratitis, unaccompanied with conjunctivitis due to any type of streptococcus. The patients, children, were otherwise healthy and gave a negative reaction to tuberculin. Streptococci could not be found in the corneal lesion, but the patients exhibited positive reactions to streptococcic allergens.

H. F. CARRASQUILLO.

General

INFECTIONS OF THE EYES. A. SORSBY, Brit. M. J. 1: 494 (April 7) 1945.

At a meeting of the Fever Group of the Society of Medical Officers of Health, London, March 9, 1945, a discussion on the epidemiology and treatment of some ocular infections was opened by Sorsby.

He said that a provisional analysis of 9,622 certificates of blindness in the L. C. C. and Middlesex areas had shown that 1,427 persons or 15 per cent were blinded by infectious diseases. In approximately three quarters of this 15 per cent ophthalmia neonatorum and syphilis were the responsible factors, ophthalmia neonatorum being the cause of blindness in 265, congenital syphilis in 516 and acquired syphilis in 290. In the remainder of the 15 per cent the factors contributing to blindness were largely trachoma (134 persons) and the various specific fevers (103 persons). The low incidence of infectious disease among the causes of blindness in England and Wales was in striking contrast to the conditions obtaining in countries like Egypt and in the Middle East generally, where over 80 per cent of all cases of blindness were due to the acute ophthalmias and, to lesser degree, to trachoma. At one time, however, infection was the major cause of blindness in England. At the beginning of the last century smallpox was the largest single factor, and toward the end of the century it was ophthalmia neonatorum. Even as late as 1922 ophthalmia neonatorum was the cause of blindness in some 30 per cent of all children at schools for the blind; by 1942 the incidence had fallen to about 10 per cent. This decline shifted the emphasis from infection to other factors—genetic anomalies in the young and degenerative lesions in the elderly.

Though the incidence of blindness from ophthalmia neonatorum has fallen so markedly, there is no evidence that the disease itself has declined to any extent. A clearer appreciation of the cause of ophthalmia neonatorum is needed, particularly as it has now been established that in only 20 to 25 per cent of cases is the disease of gonococcic origin. In at least 10 per cent of cases it is due to virus infection, and the etiology of this and of other types needs clearer recognition. While congenital syphilis has also declined to a gratifying extent, other transmitted maternal infections are only now being recognized, toxoplasmic choroidoretinitis being an example. The decline in the incidence of trachoma in childhood has only emphasized that there is still a considerable residue of trachoma in the adult population, and these persons constitute a possible source of epidemic outbreaks.

The sulfonamide compounds and penicillin are of great help in the treatment and elimination of infectious disease of the eyes. Ophthalmia neonatorum is now easily controlled by the oral administration of the sulfonamide drugs and the local application of penicillin. The acute ophthalmias of tropical and subtropical countries also respond well to the sulfonamide drugs, while in the treatment of trachoma local application of the latter had rendered the classic methods obsolete. These new drugs are, however, likely to be effective only in the bacterial infections of the outer eye. Intraocular inflammation is rarely bacterial in origin, and such common conditions as iritis and choroiditis still need intensive study.

ARNOLD KNAPP.

EXPERIENCES WITH UNBREAKABLE CONTACT LENSES. S. VON GYÖRFFLY, Ophthalmologica **108**: 44, 1944.

One hundred patients wearing contact lenses made of an unbreakable artificial resin were observed over a period of five years. In 60 per cent of the cases the lenses were entirely satisfactory; in 20 per cent

they could be worn for a reasonable period, and in a remaining 20 per cent they could not be used. The author finds it impossible to foretell the patient's aptitude in the wearing of contact lenses and finds that the greatest difficulties are fogging, photophobia and the appearance of bubbles in the fluid.

F. H. ADLER.

General Diseases

OCULAR LEPROSY IN PANAMA. R. D. HARLEY, Am. J. Ophth. **29:** 295 (March) 1946.

Harley studied 150 cases of leprosy in Panama and found ocular involvement in 90 per cent, 13 per cent of the patients being totally blind and 41 per cent having their vision reduced to 20/200 or less. The anterior segment is by far the more commonly involved. Therapy is palliative, and the leper withstands surgical procedures on the eyes better than one would suspect.

W. S. REESE.

OBSERVATIONS ON THE EFFECT OF RIBOFLAVIN ON THE ORAL LESION AND DYSPHAGIA, AND OF RIBOFLAVIN AND BREWERS' YEAST ON DARK ADAPTATION IN A CASE OF SO-CALLED PLUMMER-VINSON SYNDROME. H. POLLAK, Brit. J. Ophth. **29:** 288 (June) 1945.

An abstract of the author's summary follows:

In a male patient admitted with a complete Plummer-Vinson syndrome (hypochromic anemia, due to iron deficiency) the angular stomatitis and dysphagia cleared up rapidly under treatment with riboflavin. A half year later, when the patient's blood picture was normal, severe impairment in dark adaptation was found. The course of dark adaptation was followed by serial tests with the Crookes instrument over a period of eight months. When the patient's performance had remained stationary for two months without treatment, a slight, but significant, improvement was recorded during treatment with riboflavin. After his dark adaptation had been allowed to fall back to the original level, the patient was treated with approximately 20 Gm. of brewers' yeast daily for four months; during this period his dark adaptation gradually returned to normal, as shown by repeated tests.

A review is given of clinical evidence suggesting the association of a deficiency in riboflavin and allied factors of the vitamin B complex with the "iron deficiency syndrome," and the involvement of a "deficiency factor" in atrophic gastritis, which is considered an etiologic factor in the present case.

This hypothesis receives support from the present study, inasmuch as the impairment in dark adaptation together with the response to treatment with yeast may be taken as reflecting a disturbance in general metabolism, which appears reversible on administration of dietary factors of the vitamin B complex.

The observations recorded here and elsewhere suggest that dietary factors of the vitamin B complex may influence visual adaptation at low illumination under the experimental conditions herein described.

W. ZENTMAYER.

SOME OCULAR COMPLICATIONS OF SCRUB TYPHUS. J. MACASKILL, Brit. J. Ophth. **29:** 537 (Oct.) 1945.

The causal organism in scrub typhus (tsutsugamushi fever) is the Rickettsia nipponica, which is believed to be transmitted to man by the larvae of a mite of the genus Trombicula. There is an incubation period of about twelve days. The site of entry may be marked by an eschar, and fever, severe toxemia, headache and prostration ensue. Diffuse glandular enlargement and a maculopapular rash are common. The mortality rate varies in different regions, ranging from 10 to 15 per cent. The essential pathologic feature is a generalized vasculitis affecting the arterioles and smaller vessels. This results in necrosis and destruction of the vessel walls, with extravasation of blood into surrounding tissues. Congestion of the conjunctiva occurs invariably in the acute phase of the disease. Subconjunctival hemorrhages (also seen in malaria), intense engorgement of the retinal veins, papilledema, retinal hemorrhages and bilateral optic nerve atrophy have all been met with.

A case is reported in which bilateral optic nerve atrophy occurred. For six weeks vision was reduced to hand movements; later visual acuity was 6/9 in each eye, and there was great depression of the periphery of the field. A toxic origin is considered as the probable cause of the optic nerve atrophy. W. ZENTMAYER.

Glaucoma

CONGENITAL GLAUCOMA FOLLOWING MATERNAL RUBELLA. DUP. GUERRY, Am. J. Ophth. **29:** 190 (Feb.) 1946.

Guerry refers to Gregg's paper on congenital cataract following rubella and reports 2 cases of congenital glaucoma in which the mothers had rubella in early pregnancy. W. S. REESE.

TANGENTIAL DISPLACEMENT OF THE IRIS IN CHRONIC GLAUCOMA. W. J. B. RIDDELL, Brit. J. Ophth. **30:** 74 (Feb.) 1946.

Riddell calls attention to a condition present in the iris in certain cases of chronic glaucoma in which the normal radial arrangement of the iris stroma is replaced by a distribution running at a tangent to the pupillary margin. The change may involve a considerable segment or even the whole iris, and when it is well marked the pupil appears to have rotated around its own axis. Of 35 patients with chronic glaucoma the appearance was present in 14 (5 men and 9 women) and absent in 21 (12 men and 9 women). It is almost certainly a stage in the atrophy of the iris which occurs in the advanced phase of chronic glaucoma of long standing, but it can be found in the early stages of the disease. The author has observed the condition in a patient with megalocornea and cataract, but only when the pupil was dilated; and it appeared to be due to atrophy or rigidity in one small area at the pupillary margin.

The article is illustrated. W. ZENTMAYER.

Injuries

STEEL FOREIGN BODIES IN THE CILIARY BODY RECEIVED IN WAR INJURIES AND THEIR REMOVAL WITH ELECTROMAGNET. N. IVANOV, Vestnik oftal. 23: 9, 1944.

In an evacuation hospital, 347 extractions of intraocular foreign bodies with the electromagnet were performed during the years 1941 to 1943, the diascleral method being the one of choice. In 8 per cent, or 28 cases, the extraction of the foreign body from the ciliary body with the magnet was successful, and in only 2 cases was it not successful. The foreign bodies penetrated into the ciliary body through the cornea in 6 cases, through the limbus in 7 cases and through the sclera in 17 cases. The average size of the foreign body was about 3 mm., and they were mostly fragments of mines, grenades and bullets. In 15 cases the extraction was done within a month; in the others, within two to three months, after the injury.

Ivanov came to the following conclusions:

1. Hemophthalmos is frequently observed in injuries of the ciliary body with steel fragments.

2. Extraction of the foreign body should be done by the diascleral method after localization by the Comberg method and the electromagnet test.

3. The incision of the sclera is made over the region of the ciliary body where the foreign body is located.

4. There is little inflammatory reaction of the eye, despite the recent extraction with the magnet and a fresh injury to the eye.

O. SITCHEVSKA.

MANAGEMENT OF INCREASED INTRAOCULAR PRESSURE FOLLOWING WAR INJURIES: FIRST COMMUNICATION. C. KALFA, Vestnik oftal. 23: 25, 1944.

It is considered that the change in the vascular-nervous apparatus of the eye is the cause of increased tension after ocular injuries. In his previous work, Kalfa established the fact that there is increased tension after injury because of disturbance in the reflex which regulates the intraocular tension. At first active hyperemia, and then passive hyperemia arises because of the irritation of the autonomic apparatus of the eye which regulates the intraocular tension. If the autonomic system of the eye is eliminated by the local use of cocaine, there is no reactive hypertony. On this basis, in cases of contusion or injuries of the eye Kalfa applied a 3 per cent cocaine ointment or gave retrobulbar injections of procaine and epinephrine. This prophylactic therapy, he believes, frequently stopped or prevented the threatening symptoms of glaucoma complicating serious injuries of the eye. Case reports illustrate the author's point of view.

O. SITCHEVSKA.

Lens

A METHOD FOR THE EXTRACTION OF DISLOCATED LENSES FROM THE VITREOUS. G. BONACCOLTO, Am. J. Ophth. 28: 1335 (Dec.) 1945.

Bonaccolto briefly discusses dislocation of the lens and suggests an operation which utilizes a conjunctival flap, sutures and peripheral

iridectomy. The incision is made with a keratome and enlarged with scissors, and the lens is removed with a loop. He reports 7 cases.

W. S. REESE.

RUBELLA CATARACT: CONGENITAL CATARACT AND OTHER DEFECTS FOLLOWING GERMAN MEASLES DURING PREGNANCY OF MOTHER. A. F. M. DEROETTH and P. B. GREENE, Northwest Med. 44: 222 (June) 1945.

DeRoetth and Greene report 2 cases of congenital cataract in infants. In addition, one of the babies had microcephaly and microphthalmos, and the other had a congenital heart lesion. The authors think that the cataracts should be extracted. The Australian committee pointed out that avian and mammalian embryonic tissues are more susceptible to infection than the adult tissues. This is the basis of Goodpasture's virus culture technic. The human embryo possibly possesses the same susceptibility to infection. The virus of rubella may pass the chorionic villi more readily than bacteria before a placental barrier has been developed. The Australian committee made the following suggestions: Expose all young girls to rubella; try to isolate the virus and prepare a vaccine against the disease; study the effect of convalescent serum, to be used on the pregnant mother in case she did not have rubella before. The question arises whether therapeutic abortion is indicated if the disease is contracted in the first three months of pregnancy.

J. A. M. A. (W. ZENTMAYER.)

METABOLISM OF THE CRYSTALLINE LENS. M. RIOS SASIAIN, Arch. Soc. oftal. hispano-am. 4: 273 (April) 1945.

After discussing thoroughly the metabolism of the lens, Sasiain reaches the following conclusions:

The metabolism of the crystalline lens is performed through electronic changes in the presence of full anoxybiosis. The presence of catalytic enzymes and oxyreducing systems are indispensable for the lenticular metabolism. Transparency and the act of accommodation are the two factors for which the metabolism of the lens is important. The reducing power of the lens must reach a certain level, which must be kept constant in order that transparency and accommodation be present. When, owing to various causes, the metabolic harmony of the integrating elements of the crystalline lens is lost, there is precipitation of these elements, and opacification of the lens results.

H. F. CARRASQUILLO.

Methods of Examination

REPEATABILITY OF KERATOMETRIC READINGS. R. E. BANNON and R. WALSH, Am. J. Ophth. 29: 76 (Jan.) 1946.

Bannon and Walsh conclude that this study of the repeatability of keratometric readings on 100 eyes indicates that the keratometer is a reliable instrument and that the importance of the interpretation of individual examiners is negligible.

W. S. REESE.

STEREOSCOPIC OPHTHALMOSCOPY WITH FOCAL ILLUMINATION. J. MALBRAN, Arch. de oftal. de Buenos Aires **19:** 455 (Nov.) 1944.

The author gives a brief historical review of all the methods of ophthalmoscopy and deals fully with the new method devised by Goldmann. In this method observation of the eyeground is made with the slit lamp of Haag and Streit and prisms which reduce to 5 degrees the angle formed by the luminous rays and the axis of the corneal microscope. It is also necessary to employ a contact lens which in certain respects differs from that devised by Koeppe. The advantages of the method are mentioned.

H. F. CARRASQUILLO.

Neurology

UNILATERAL LACRIMATION ASSOCIATED WITH CHEWING: REPORT OF A CASE. I. B. BENDER, Am. J. Orthodontics **31:** 692 (Nov.) 1945.

Bender reports a case in which stimulation of the area of the anterior palatine pad produced only congestion, lacrimation and pain. The effects were best produced by rubbing with a finger or by pressure of a denture during mastication. While the symptoms could be produced by reading small print and by blowing the nose, the dental factor was the more important. The pad area was definitely localized as the trigger point by the fact that unilateral biting and movements of the muscles of mastication did not reproduce the syndrome. When there is independent biting either on the right or on the left side, there is no compression or rubbing on the pad area. With normal chewing the force of mastication produces a pressure because of the closeness of adaptation of the dentures on the yielding mucosal tissues. The present case differs from those reported in the literature in that congestion and pain were associated with lacrimation. Whether this can be attributed to stimulation of both the autonomic and the sensory system is difficult to say.

J. A. M. A. (W. ZENTMAYER.)

DENIAL OF BLINDNESS BY PATIENTS WITH CEREBRAL DISEASE. F. D. REDLICH and J. F. DORSEY, Arch. Neurol. & Psychiat. **53:** 407 (June) 1945.

Redlich and Dorsey observed 6 instances of denial of blindness over a period of eighteen months in a 600 bed hospital. Most examiners are reluctant to make any attempt to stress forcibly to the patient such a severe defect as blindness. All the patients who present such a syndrome are deteriorated and have disturbances of retention and orientation, hallucinations and delusions. The syndrome of denial of their own blindness in the authors' series was caused by a diabetic retinopathy in 1 patient, by atrophy of the optic nerve in another patient and by bilateral hemianopsia due to tumor or to vascular lesions in 4 patients. All these patients had diffuse cerebral lesions. All showed intellectual deterioration: disorientation; severe impairment of recent memory and retention, and confabulation. The existence of bilateral focal lesions of the visual radiations or of the occipital visual areas leading to bilateral hemianopsia seems to play an important part in the pathogenesis of the syndrome. The interruptions of reverberating circuits between the thalamus and the sensory cortex constitute the most prominent etiologic factor.

J. A. M. A. (W. ZENTMAYER)

INDIRECT INJURY OF THE OPTIC CHIASMA: A Case Report. E. B. C. HUGHES, Brit. J. Ophth. **29:** 629 (Dec.) 1945.

A man aged 45 sustained a severe craniocerebral injury by striking the front of the head against a windshield. Examination one month later revealed no scalp wound, but there was severe bruising of both orbits and the frontal region. Roentgenograms showed a horizontal fissured fracture running above the frontal sinuses. On the right side this extended just over the midline and then ran vertically downward into the right frontal sinus; on the left side it ran as far laterally as the outer margin of the orbit and then extended vertically downward onto the roof of the orbit. There were complete bilateral anosmia, complete paralysis on the right side of the face with loss of taste and mild hemiparesis with increases of tone and of the tendon reflexes.

Both optic disks were abnormally pale, but the retinal vascular tree was normal. Visual acuity was 6/9 in each eye. The visual fields showed complete loss of the temporal field with involvement of fixation. The nasal fields were normal. Surgical exploration showed that the right optic nerve from the optic canal to its junction with the chiasm was normal. The left side of the chiasm and the left optic nerve were partially obscured by recent, grayish yellow, filmy adhesions; these were easily brushed away. The left optic nerve appeared normal. No gross injury of the chiasm could be seen. Four months later no changes had taken place in the optic nerves or the visual fields.

Hughes accepts the theory put forward by Traquair, Dott and Russell that the lesion is due to damage of the small vessels of the chiasm, most probably from a stretch injury.

The article is illustrated.

W. ZENTMAYER.

Operations

DACRYO-CYSTO-RHINOSTOMY. T. K. LYLE, A. G. GROSS, J. F. SIMPSON and G. A. FRASER, Brit. J. Ophth. **30:** 102 (Feb.) 1946.

A description is given of a technic employed in dacryocystorhinostomy. The records of 56 cases in which the operation was performed are reproduced. The cases are analyzed to show the satisfactory results obtained with this procedure, even in cases in which previous surgical intervention has been carried out. The technic of the operation is shown by a number of adequate illustrations.

W. ZENTMAYER.

USE OF CAUTERY IN PLASTIC OPERATION ON THE EYELIDS. D. SIMPSON BRISTOL, Brit. M. J. **2:** 424 (Sept. 29) 1945.

The author used the electric cautery alone or in combination with the scalpel, though the major part of the work was done with the cautery. The conditions dealt with were ectropion, entropion, symblepharon, distichiasis and ptosis. The aim was to produce enough contracture by scarring of the underlying tissue to correct the deformity of the lids. The ordinary Weiss cautery was used, and the size of the point varied from 5 mm. to 1 cm. in length and 2 to 0.5 mm. in width at the base.

After anesthesia, entropion and ectropion were treated by introducing the red-hot point through the skin, or through the mucous membrane, down to the tarsal plate five or six times along the whole length of the lid. The point is held in each puncture about three seconds. If the condition is old and fairly prominent, it is necessary to excise a longitudinal strip of conjunctiva, of about one-third the width of the exposed conjunctiva. The treatment has to be thorough as an overeffect is not likely to follow.

Treatment of ectropion of the upper lid should be particularly thorough. The middle third of the conjunctiva is resected along the whole length of the lid, and then the red-hot point is plunged deep into the connective tissue, to and beyond the tarsal plate, at a number of places.

Symblepharon is a difficult condition. The author has attempted to disengage adhesions when the cornea was invaded. The cautery is applied to divide the adhesions at the most suitable spot, the point being carried down to the sclera. Each adhesion is treated in this way. The adhesions seldom reunite.

In treatment of distichiasis the point of the cautery is pushed down each hair follicle in turn and retained for two seconds. The lashes do not grow again, and no damage is done to the lid margins.

In cases of congenital ptosis in children the author has attempted to cause the levator muscle to form fresh adhesions to the tarsal plate. A row of six punctures is made just above the tarsal plate, and then each puncture is entered again, the point being passed upward and in the subcutaneous tissues as far as possible. The point should be 1.5 cm. long.

The author believes that this is a very useful procedure and that its simplicity is not generally recognized. In his own practice the results have been superior to those obtained with scalpel and stitch.

ARNOLD KNAPP.

Orbit, Eyeball and Accessory Sinuses

OBSERVATION ON SO-CALLED THYROTROPIC EXOPHTHALMOS. H. ZONDEK and A. TICHO, Brit. M. J. **1**: 836 (June 16) 1945.

Another form of exophthalmos than the one usually appearing with exophthalmic goiter has recently been described as attributable to the thyrotropic factor of the anterior lobe of the pituitary gland. This form goes under the name of thyrotropic exophthalmos.

In thyrotoxic exophthalmos the following factors are considered important from the etiologic point of view: (1) contraction of Müller's palpebral muscles, owing to increased sympathetic stimulation; (2) adynamia of the rectus muscles, which normally retract the eyeball; (3) occurrence of anatomic changes, which are generally not well marked. The extrabulbar muscles are of normal shape and consistency, though in some cases general wasting of the muscle fibers is present.

In thyrotropic exophthalmos the proptosis is more important in the development of the exophthalmos than is the widening of the palpebral fissure. The most important cause of the proptosis seems to be a muscular disorder, such as (1) diffuse extensive fibrosis, (2) edema, (3) abundant round cell infiltration or (4) a special type of degeneration

resulting in fibrosis or absorption of muscle fibers (Mulvany). In thyrotropic exophthalmos the proptosis is generally greater than in the thyrotoxic form and corresponds more closely to the malign form of the thyrotoxic variety.

If the mechanical theory for the production of the exophthalmos does not hold true in all cases, its coincidence with diabetes insipidus, a symptom which is more or less characteristic of this disease, would indicate involvement of the pituitary gland or the diencephalic nuclei.

The authors report on the examination in 3 cases of excessive exophthalmos which are recorded as instances of thyrotropic exophthalmos. The ophthalmic manifestations were particularly severe in all these cases. In 2 cases pronounced decalcification of the bones of the skull and enlargement of the sella turcica were present; this sign points to involvement of the pituitary-diencephalic region. In both cases dextrose tolerance curves of diabetic type were obtained. The abnormally high cholesterol level in the blood in 2 cases argues against hyperthyroidism. In all 3 cases the disease responded favorably to diiodotyrosine. In 1 case (postoperative exacerbation of the exophthalmos) administration of diiodotyrosine, in combination with irradiation of the pituitary body on two occasions, resulted in considerable improvement of the eyes (regresson of proptosis and disappearance of chemosis and epiphora) and thus rendered surgical intervention unnecessary. In view of the fact that the part played by the thyrotropic hormone in the production of this type of exophthalmos is doubtful, whereas the role of the pituitary-diencephalic system seems to be highly probable, the authors suggest the looser definition of "pituitary-diencephalic exophthalmos."

ARNOLD KNAPP.

Parasites

OCULAR GNATHOSTOMIASIS. K. SEN, Brit. J. Ophth. 29: 618 (Dec.) 1945.

The gnathostoma was first recovered from the stomach nodule of a tiger by Owen; since then, it has been recovered from the stomach nodules of domestic cats, wild cats, leopards and dogs and from esophageal nodules in weasels in the Malay States, China, India and Japan. The first case of human infection was reported by Levinson in 1890. The present case is probably the first reported instance of this worm in a human eye.

A case of gnathostoma in the anterior chamber of the eye of a Hindu Brahmin aged 26 is described. The onset was with a dull, aching pain on the left side of the nose, which extended to the left frontal and temporal regions, followed by swelling of the face, extending from the angle of the mouth to the roots of the hair. These symptoms were followed by orbital cellulitis and vitreous and retinal hemorrhages. There occurred two attacks of iritis with the development of gray nodules on the iris, which completely disappeared, leaving gray depressions on the iris. Later, two more attacks of iritis occurred, with the formation of a single pigmented nodule on the iris in each attack. The nodule completely disappeared, leaving no mark after the first attack.

In the second attack, which was more severe, the pigmented nodule was seen to be the worm. The worm was removed at operation, and the eye made an uneventful recovery, though optic nerve atrophy developed.

It was not clear how the worm came to the side of the nose from the stomach without causing any signs or symptoms. The probable area of its entry into the eyeball from the orbit could be seen as a scar just below the macula.

The article is illustrated. W. ZENTMAYER.

Trachoma

ETIOLOGY OF TRACHOMA. P. DE GÓES, Rev. med. brasil. **18:** 61, 1945.

The author reviews the literature on the etiology of trachoma. He discusses the nosonomic concept of the disease and expresses the belief that it is a specific disease. The part played by constitution and other intrinsic causes which greatly influence the appearance and evolution of the disease are discussed. Various agents are considered as causative factors in the disease. Noguchi's work is studied in detail, the author believing that Noguchi's granulosis represents an incubated germ of the disease. He believes that the rickettsias described by Busacca represent elementary corpuscles of the virus of the disease. As to the Halberstaedter-Prowazek corpuscles, the author believes that they represent a special stage of the virus. The virus causation is discussed, this theory being, in the author's opinion, the most probable.

M. E. ALVARO.

Toxic Amblyopia

QUININE AMBLYOPIA WITH SPONTANEOUS DETACHMENT. J. L. HART, Brit. J. Ophth. **29:** 375 (July) 1945.

After the administration of quinine for a febrile condition the patient was delirious, with hallucinations, and claimed to be totally blind. The pupils were semidilated but reacted both directly and consensually. The eyes blinked when the hand was moved before the face. Care was taken to avoid an air-produced corneal reflex. In the right eye all but the temporal half of the disk was blurred. The vessels were scarcely visible at a distance from the disk. In the left eye the disk was pale. Eleven days after the onset the retina of the left eye was seen to be detached above the nerve head. On the twenty-first day vision in the right eye was limited to counting fingers at 2 meters and in the left eye to mere ability to count fingers. The patient received between 60 and 90 grains (0.39 to 0.585 Gm.) of quinine. W. ZENTMAYER.

Book Reviews

Augenärztliche Eingriffe. Fifth edition. By Professor J. Meller and Dr. J. Böck, with additions by K. Kofler and A. Pillat. Pp. 460, with 267 illustrations. Vienna: Julius Springer, 1946.

The fourth edition of this much enlarged and revised textbook appeared in 1938 and was reviewed in these ARCHIVES (**21**: 561 [May] 1939). That edition was so popular that a new one soon became necessary and was ready in October 1944, but the terrible and tragic days which the war has brought to Austria, and particularly to Vienna, postponed its publication to this year. The excellent qualities of the fourth edition are just as outstanding in the fifth. The operations described are, with a few exceptions, those which have stood the test of time at the First Ophthalmic Clinic of the Allgemeines Krankenhaus in Vienna.

The young ophthalmic surgeon will not find the description of the fundamental principles in ophthalmic surgery so thoroughly treated and so well explained in any other book. In many cases only one method of operating is selected, as Meller says it is not the method itself but the experience with it which is the determining factor in success. When one method is not sufficient for all types of cases, such as those of glaucoma, a number of operations are described. The chapter on muscle surgery does not include the methods which are in general practice in this country. Corneal transplantation is not described and is apparently not practiced in the Vienna clinic. In the chapter on retinal detachment two additions are noted. The first is the return to Gonin's original method with the thermocautery when there is a vitreous pull from the radially placed operculum and when attempts with the high frequency current have failed to close a retinal hole. The second addition is the operation for shortening of the eyeball by scleral resection according to the method now practiced by Lindner. This method is suited for desperate cases of detachment in which no tear can be found, and especially in aphakic eyes or when other procedures have failed. A number of operations for glaucoma are described, but it is not clear which one the authors think the best in certain circumstances. The discussion of the intracapsular method of cataract extraction has been revised and has received a much more complete treatment at the hands of the co-editor, J. Böck, and the various steps and the difficulties of the operation are fully outlined. At the same time, the book teaches that the beginner in ophthalmic surgery should first master the difficulties of the extracapsular technic and the intracapsular method should be selected for the cases in which this operation is superior.

Professor Meller closes his foreword with a dedication of farewell. Even if he withdraws from the active editorship of this book, future editions will carry his method of exposition of the principles of ophthalmic surgery and be of lasting value to every young ophthalmologist. An English translation, which is on the way, will be eagerly welcomed by many new readers.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 17 Horseferry Rd., London, England.

Pan-American Association of Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretaries: Dr. Conrad Berens, 35 E. 70th St., New York. Dr. M. E. Alvaro, 1511 Rua Consolacão, São Paulo, Brazil.
Place: Habana, Cuba. Time: Feb. 22-28, 1948.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. W. Clark Souter, 9 Albyn Pl., Aberdeen, Scotland.
Secretary: Dr. Frederick Ridley, 12 Wimpole St., London, W. 1.

Chengtu Ophthalmological Society

President: Dr. Eugene Chan.
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China.
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd. Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

Faculty of Ophthalmologists

President: Brig. Sir Stewart Duke-Elder, 63 Harley St., London, W. 1, England.
Secretary: Mr. Frank W. Law, 45 Lincoln's Inn Fields, London, W. C. 2, England.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
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Hungarian Ophthalmological Society

President: Prof. I. Imre, Budapest.
Assistant Secretary: Dr. Stephen de Grósz, University Eye Hospital, Máriautca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: Mr. T. Harrison Butler, 61 Newhall St., Birmingham 3, England.
Place: Birmingham and Midland Eye Hospital.

North of England Ophthalmological Society

President: Mr. E. F. Wilson, 24 Upper Northgate St., Chester.
Secretary: Mr. William M. Muirhead, 70 Upper Hanover St., Sheffield 3.
Place: Manchester, Leeds, Newcastle-upon-Tyne, Liverpool, Sheffield and Bradford, in rotation. Time: October to May.

Ophthalmological Society of Australia

President: Dr. J. Ringland Anderson, Astor House, 108 Collins St., Melbourne, Victoria.
Secretary: Dr. D. A. Williams, 27 Commonwealth St., Sydney.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the secretary, Dr. Mohammed Khalil.

Ophthalmological Society of Hospital de Nuestra Señora de la Luz

Chairman: Dr. Manuel J. Icaza y Dublán, México, D. F., Mexico.
Secretary: Dr. Jorge Meyrán, México, D. F., Mexico.

Ophthalmological Society of South Africa

President: Dr. A. W. Sichel, National Mutual Bldg., Church Square, Cape Town.
Secretary: Dr. J. K. de Kock, Groote Kerk Bldg., 32 Parliament St., Cape Town.

Ophthalmological Society of the United Kingdom

President: Dr. A. J. Ballantyne, 11 Sandyford Pl., Glasgow C. 3, Scotland.
Secretary: Mr. J. H. Doggart, 49 Wimpole St., London, W. 1.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Rd., Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Mr. F. A. Williamson-Noble, 27 Harley St., London, W. 1, England.
Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Place: Oxford. Time: July 3-5, 1947.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
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Philippine Ophthalmological and Otolaryngological Society

President: Dr. Antonio S. Fernando, Philippine General Hospital, Manila.
Secretary-Treasurer: Dr. José N. Cruz, Philippine General Hospital, Manila.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Puerto Rico Medical Association, Section of Ophthalmology and Otolaryngology

President: Dr. José T. Picó, Avenida Ponce de Leon 15, San Juan 10.
Secretary: Dr. Ricardo F. Fernández, Avenida Ponce de Leon 15, San Juan 10.

Revista Brasileira de Oftalmologia

President: Dr. Caldas Britto, Largo de Carioca 5-6° andar, Rio de Janeiro, Brazil.
Secretary: Dr. Evaldo Campos, R. Rodrigo Silva 7-1° andar, Rio de Janeiro, Brazil.
Place: Rio de Janeiro, Brazil. Time: Third Friday of every month from April to December.

Royal Society of Medicine, Section of Ophthalmology

President: Col. F. A. Juler, 96 Harley St., London, W. 1, England.
Secretary: Dr. Harold Ridley, 60 Queen Anne St., London, W. 1, England.

São Paulo Society of Ophthalmology

President: Dr. Armando Gallo, Rua Benjamin Constant, 67, 9° Andar São Paulo, Brazil.
Secretary: Dr. F. Amédée Féret Filho, Rua Marconi, 48, 4° Andar, São Paulo. Brazil.

Scottish Ophthalmological Club

President: Dr. S. Spence Meighan, 13 Woodside Pl., Glasgow, C. 3.
Secretary: Dr. Alexander Garrow, 15 Woodside Pl., Glasgow, C. 3.
Place: Edinburgh and Glasgow, in rotation.

Sociedad Argentina de Oftalmologia

Chairman: Dr. Jorge Malbrán, Buenos Aires.
Secretary: Dr. Benito Just Tiscornia, Santa Fe 1171, Buenos Aires.

Sociedad Cubana de Oftalmologia

President: Prof. Lorenzo Comas, Calle D #461, Vedado, Habana, Cuba.
Secretary: Dr. Oscar F. Horstmann, Calle 5ta. #702, Vedado, Habana, Cuba.
Time: Second Tuesday of every month.

Sociedad Oftalmologia del Litoral, Rosario (Argentina)

President: Prof. Dr. Carlos Weskamp, Laprida 1159, Rosario.
Secretary: Dr. Arturo Etchemendigaray, Villa Constitución, Santa Fé.
Place: Rosario. Time: Last Saturday of every month, April to November. All correspondence should be addressed to the President.

SOCIEDADE DE OFTALMOLOGÍA DEL NORTE

President: Dr. Alberto Cárdenas.
Secretary: Dr. Jorge Luis Castillo, Mendoza 421, Tucumán, Argentina.

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAES

President: Prof. Hilton Rocha, Rua Rio de Janeiro 2251, Bello Horizonte, Minas Geraes, Brazil.
Secretary: Dr. Ennió Coscarelli, Rua Aimorés 1697, Bello Horizonte, Minas Geraes, Brazil.

SOCIEDADE DE OFTALMOLOGIA E OTORINOLARINGOLOGIA DO RIO GRANDE DO SUL

President: Dr. Luiz Assumpção Osorio, Edificio Vera Cruz, Apartamento 134, Porto Alegre, Rio Grande do Sul.
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul.

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DO BAHIA

President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil.
Secretary: Dr. Adroaldo de Alencar, Brazil.
All correspondence should be addressed to the President.

SOCIETÀ OFTALMOLOGICA ITALIANA

President: Prof. Dott. Giuseppe Ovio, Ophthalmological Clinic, University of Rome, Rome.
Secretary: Prof. Dott. Epimaco Leonardi, Via del Gianicolo, 1, Rome.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

Secretary: Dr. K. O. Granström, Södermalmstorg 4, Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Derrick Vail, 55 E. Washington St., Chicago.
Secretary: Dr. R. J. Masters, 23 E. Ohio St., Indianapolis.
Place: Atlantic City. Time: June 9-13, 1947.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Alan C. Woods, Johns Hopkins Hospital, Baltimore 5.
President-Elect: Dr. C. H. McCaskey, 20 N. Meridian St., Indianapolis, Ind.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100–1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago. Time: Oct. 12-17, 1947.

AMERICAN OPHTHALMOLOGICAL SOCIETY

President: Dr. John W. Burke, 1740 M St. N. W., Washington, D. C.
Secretary-Treasurer: Dr. Walter S. Atkinson, 129 Clinton St., Watertown, N. Y.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

Chairman: Dr. Derrick Vail, 55 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Brittain F. Payne, 17 E. 72d St., New York.
Assistant Secretary-Treasurer: Dr. Hunter Romaine, 35 E. 70th St., New York.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

President: Dr. Alexander E. MacDonald, 170 St. George St., Toronto 5.
Secretary-Treasurer: Dr. L. J. Sebert, 170 St. George St., Toronto 5.

CANADIAN OPHTHALMOLOGICAL SOCIETY

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Secretary-Treasurer: Dr. Kenneth B. Johnston, Suite 1, 1509 Sherbrooke St. W., Montreal.

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

President: Mr. Mason H. Bigelow, 1790 Broadway, New York.
Secretary: Miss Regina E. Schneider, 1790 Broadway, New York.
Executive Director: Mrs. Eleanor Brown Merrill, 1790 Broadway, New York.

SECTIONAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Anthony Ambrose, 31 Lincoln Park, Newark.
Secretary: Dr. William F. Keim Jr., 15 Washington St., Newark 2.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Walter Stevenson, 510 Maine St., Quincy, Ill.
Secretary-Treasurer: Dr. William F. Hubble, 866 Citizens Bldg., Decatur, Ill.

CENTRAL WISCONSIN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. P. G. Spelbring, 131 S. Barstow St., Eau Claire.
Secretary: Dr. G. L. McCormick, 650 S. Central Ave., Marshfield.

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. J. Pinkerton, 7 Young Hotel Bldg., Honolulu.
Secretary-Treasurer: Dr. L. Q. Pang, 52 S. Vineyard St., Honolulu 39.
Place: Honolulu. Time: Third Thursday of each month.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. Howard F. Hill, 177 Main St., Waterville, Maine.
Secretary-Treasurer: Dr. Merrill J. King, 264 Beacon St., Boston 16.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. D. H. O'Rourke, 1612 Tremont Pl., Denver.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Julius A. Weber, 640 Stimson Bldg., Seattle 1, Wash.
Secretary-Treasurer: Dr. Barton E. Peden, 301 Stimson Bldg., Seattle 1, Wash.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month except June, July and August.

ROCK RIVER VALLEY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. Sheldon Clark, Sterling, Ill.
Secretary-Treasurer: Dr. Harry R. Warner, 321 W. State St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Tiedke, 120 N. Michigan Ave., Saginaw, Mich.
Secretary-Treasurer: Dr. James Curts, 330 S. Washington Ave., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July, August and September.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. J. C. Decker, 515 Francis Bldg., Sioux City, Iowa.
Secretary-Treasurer: Dr. J. E. Dvorak, 408 Davidson Bldg., Sioux City, Iowa.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Savage Zerfoss, 165–8th Ave. N., Nashville 3, Tenn.
Secretary: Dr. Alston Callahan, 908 S. 20th St., Birmingham 5, Ala.

SOUTHWESTERN ACADEMY OF EYE, EAR, NOSE AND THROAT

President: Dr. H. L. Brehmer, 221 W. Central Ave., Albuquerque, N. Mex.
Secretary: Dr. A. E. Cruthirds, 1011 Professional Bldg., Phoenix, Ariz.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. M. Dodge, 716 First National Bank Bldg., Battle Creek.
Secretary-Treasurer: D. Kenneth Lowe, 25 W. Michigan Ave., Battle Creek.
Time: Last Thursday of September, October, November, March, April and May.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. J. V. Foster, State College.
Secretary-Treasurer: Dr. J. McClure Tyson, Deposit National Bank Bldg., Dubois.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. E. C. Moulton, 619 Garrison Ave., Fort Smith.
Secretary: Dr. K. W. Cosgrove, 7 Urquhart Bldg., Little Rock.

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. George H. Stine, 23 E. Pikes Peak Ave., Colorado Springs.
Secretary: Dr. J. Leonard Swigert, 320 Republic Bldg., Denver.
Place: University Club, Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Paul B. MacCready, 442 Temple St., New Haven.
Secretary-Treasurer: Dr. W. H. Turnley, 1 Atlantic St., Stamford, Conn.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William O. Martin Jr., Doctors Bldg., Atlanta.
Secretary-Treasurer: Dr. C. K. McLaughlin, 666 Cherry St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. W. E. Stewart, 721 Wabash Ave., Terre Haute.
Secretary: Dr. Russell A. Sage, 23 E. Ohio St., Indianapolis.
Place: French Lick. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. S. O'Brien, University Hospital, Iowa City.
Secretary-Treasurer: Dr. Carl A. Noe, 120–3d Ave. S. E., Cedar Rapids.

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. Granger, Emporia.
Secretary: Dr. George F. Gsell, 911 Beacon Bldg., Wichita 2.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. George S. Adkins, 121 N. President St., Jackson, Miss.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

MEDICAL SOCIETY OF THE STATE OF PENNSYLVANIA, SECTION ON EYE, EAR, NOSE AND THROAT DISEASES

Chairman: Dr. William T. Hunt Jr., 1205 Spruce St., Philadelphia 7.
Secretary: Dr. Gabriel Tucker, 250 S. 18th St., Philadelphia 3.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Edmond L. Cooper, 1553 Woodward Ave., Detroit 26.
Secretary: Dr. Ralph H. Gilbert, 110 Fulton St. E., Grand Rapids.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Karl C. Wold, 1051 Lowry Bldg., St. Paul 2.
Secretary: Dr. William A. Kennedy, 372 St. Peter St., St. Paul 2.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. H. Casebeer, 44 W. Park Ave., Butte.
Secretary: Dr. Fritz D. Hurd, 309 Medical Arts Bldg., Great Falls.

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. John Peterson, 1307 N St., Lincoln.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. C. W. Buvinger, 50 Washington St., East Orange.
Secretary: Dr. Z. Laurence Griesemer, 1145 E. Jersey St., Elizabeth.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Maxwell D. Ryan, 660 Madison Ave., New York 21.
Secretary: Dr. Thomas H. Johnson, 30 W. 59th St., New York.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. A. J. Ellington, 412 S. Spring St., Burlington.
Secretary: Dr. J. A. Harrill, Bowman Gray School of Medicine, Winston-Salem.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. D. Perrin, 221–5th St., Bismarck.
Secretary-Treasurer: Dr. M. T. Lampert, Minot.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Wilfred Belnap, 833 S. W. 11th Ave., Portland.
Secretary-Treasurer: Dr. C. W. Kuhn, 1020 S. W. Taylor St., Portland 5.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Thomas F. Furlong Jr., 36 Parking Plaza, Ardmore.
Secretary: Dr. Benjamin F. Souders, 143 N. 6th St., Reading.
Time: Last week in April.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society, Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Ruskin Anderson, 145 N. Converse St., Spartanburg.
Secretary-Treasurer: Dr. Roderick Macdonald, 330 E. Main St., Rock Hill.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George Burchfield, Maryville.
Secretary-Treasurer: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. H. Rosebrough, 603 Navarro St., San Antonio.
Secretary: Dr. M. K. McCullough, 1717 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. E. B. Fairbanks, 315 Medical Arts Bldg., Salt Lake City.
Secretary-Treasurer: Dr. Dean Spear, 516 Boston Bldg., Salt Lake City.
Place: University Club, Salt Lake City. Time: 7:00 p. m., third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Thomas E. Hughes, 1000 W. Grace St., Richmond.
Secretary-Treasurer: Dr. Francis H. McGovern, 105 S. Union St., Danville.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. E. L. Mather, 39 S. Main St., Akron, Ohio.
Secretary-Treasurer: Dr. V. C. Malloy, 2d National Bank Bldg., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. B. M. Cline, 153 Peachtree St. N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 815 Doctors Bldg., Atlanta, Ga.
Place: Academy of Medicine. Time: 7:30 p. m., fourth Monday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Jonas Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. Fred Reese, 330 N. Charles St., Baltimore 1.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to March.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. W. Chunn Parsons, 425 Woodward Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. Benjamin C. Rosenthal, 140 New York Ave., Brooklyn.
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Ave., Brooklyn 13.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. William M. Howard, 389 Linwood Ave., Buffalo 9.
Secretary-Treasurer: Dr. Sheldon B. Freeman, 196 Linwood Ave., Buffalo 9.
Time: Second Thursday of each month from October to May.

Central New York Eye, Ear, Nose and Throat Society

President: Dr. Uri Doolittle, University Bldg. Syracuse.
Secretary-Treasurer: Dr. Alfred W. Doust, 306 State Tower Bldg., Syracuse.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. Douglas Chamberlain, Providence Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. W. A. Mann, 30 N. Michigan Ave., Chicago 2
Secretary: Dr. J. R. Fitzgerald, 3215 W. North Ave., Chicago.
Place: Continental Hotel, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati General Hospital Ophthalmology Staff

Chairman: Dr. D. T. Vail, 441 Vine St., Cincinnati.
Secretary: Dr. A. A. Levin, 441 Vine St., Cincinnati.
Place: Cincinnati General Hospital. Time: 7:45 p. m., third Friday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. H. H. Wygand, 624 Guardian Bldg., Cleveland.
Time: Second Tuesday in October, December, February and April.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Burton Chance, 317 S. 15th St., Philadelphia.
Clerk: Dr. George F. J. Kelly, 37 S. 20th St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

COLUMBUS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

Chairman: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. J. Miller, 21 E. State St., Columbus, Ohio.
Place: University Club. Time: 6:15 p. m., first Monday of each month, from October to May, inclusive.

CORPUS CHRISTI EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. L. W. O. Jansen, 710 Medical Professional Bldg., Corpus Christi, Texas.
Secretary: Dr. F. B. Kelly, 519 Medical Professional Bldg. Corpus Christi, Texas.
Time: 6:30 p. m., third Tuesday of each month from October to May.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Speight Jenkins, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. L. Darrough, Dallas Medical and Surgical Clinics, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. I. McPherrin, 406–6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. C. C. Jones, Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., fourth Monday of every month from September to May.

DETROIT OPHTHALMOLOGICAL CLUB

Chairman: Members rotate alphabetically.
Secretary: Dr. Wesley G. Reid, 974 Fisher Bldg., Detroit 2.
Place: Club rooms of Wayne County Medical Society. Time: First Monday of each month, November to April, inclusive.

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Bruce Fralick, 201 S. Main St., Ann Arbor, Mich.
Secretary: Dr. William S. Gonne, 619 David Whitney Bldg., Detroit 26.
Place: Club rooms of Wayne County Medical Society. Time: 6:30 p. m., third Thursday of each month from November to April, inclusive.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. Frank C. Furlong, 713 Union St., Schenectady.
Secretary-Treasurer: Dr. E. Martin Freund, 762 Madison Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. R. Lees, 602 W. 10th St., Fort Worth 2, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1305 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION

President: Dr. J. Matt Robison, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. John H. Barrett, 1304 Walker Ave., Houston, Texas.
Place: River Oaks Country Club. Time: 6:30 p. m., second Thursday of each month from October to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. Jerome Littell, 603 Hume Mansur Bldg., Indianapolis.
Secretary: Dr. J. Lawrence Sims, 23 E. Ohio St., Indianapolis.
Place: Indianapolis Athletic Club. Time: 6:30 p. m., second Thursday of each month from November to May.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Edgar Johnson, 906 Grand Ave., Kansas City, Mo.
Secretary: Dr. W. E. Keith, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Robert G. Thornburgh, 117 E. 8th St., Long Beach 2, Calif.
Secretary-Treasurer: Dr. Kirt Parks, 605 Professional Bldg., Long Beach 2, Calif.
Place: Seaside Hospital. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William D. Donoher, Los Angeles.
Secretary-Treasurer: Dr. K. C. Brandenburg, 110 Pine Ave., Long Beach 2, Calif.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE AND EAR SOCIETY

President: Dr. Joseph S. Heitger, Heyburn Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. J. W. Fish, 321 W. Broadway, Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

LOWER ANTHRACITE EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Each member in alphabetical order.
Secretary: Dr. James J. Monohan, 31 S. Jardin St., Shenandoah, Pa.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. P. S. Constantinople, 1835 I St. N. W., Washington.
Secretary: Dr. Frazier Williams, 1801 I St. N. W., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month from September to May.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. Ralph T. Rank, 238 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. Frank G. Treskow, 411 E. Mason St., Milwaukee 2.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month from October to May.

Montgomery County Medical Society

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel: Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. F. Badeaux, 502 Cherrier St., Montreal, Canada.
Secretary: Dr. John V. V. Nicholls, 1414 Drummond St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. M. M. Cullom, 700 Church St., Nashville, Tenn.
Secretary: Dr. R. E. Sullivan, 432 Doctors Bldg., Nashville 3, Tenn.
Place: James Robertson Hotel. Time: 6:30 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Secretary: Dr. Mercer G. Lynch, 1018 Maison Blanche Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to May.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Brittain F. Payne, 17 E. 72d St., New York 21.
Secretary: Dr. Milton Berliner, 57 W. 57th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Benjamin Friedman, 6 W. 77th St., New York.
Secretary: Dr. Benjamin Esterman, 983 Park Ave., New York 28.
Place: New York Academy of Medicine, 2 E. 103d St. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Oklahoma City Academy of Ophthalmology and Otolaryngology

President: Dr. S. R. Shaver, 117 N. Broadway, Oklahoma City.
Secretary: Dr. William Mussil, Medical Arts Bldg., Oklahoma City.
Place: University Hospital. Time: Second Tuesday of each month from September to May.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. A. A. Steinberg, 1502 Farnam St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha 2.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m. dinner; 7 p. m. program; third Wednesday of each month from October to May.

Passaic-Bergen Ophthalmological Club

President: Dr. Thomas Sanfacon, 340 Park Ave., Paterson, N. J.
Secretary-Treasurer: Dr. J. Averbach, 435 Clifton Ave., Clifton, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

President: Dr. L. Waller Winckler, Philadelphia.
Secretary: Dr. Robert T. M. Donnelly, 255 S. 17th St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Jay G. Linn, Jenkins Arcade, Pittsburgh.
Secretary: Dr. Robert J. Billings, Jenkins Arcade, Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Reading Eye, Ear, Nose and Throat Society

President: Dr. Claude W. Bankes, 212 N. 6th St., Reading, Pa.
Secretary: Dr. Paul C. Craig, 232 N. 5th St., Reading, Pa.
Place: Wyomissing Club. Time: 6:30 p. m., third Wednesday of each month from September to July.

Richmond Eye, Ear, Nose and Throat Society

President: Dr. Luther C. Brawner, Professional Bldg., Richmond, Va.
Secretary: Dr. Clifford A. Folkes, Professional Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Frank Barber, 75 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.

St. Louis Ophthalmic Society

President: Dr. A. Lange, 3903a Olive St., St. Louis.
Secretary: Dr. William Kleinberg, Frisco Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Fourth Friday of each month from October to April, inclusive, except December, at 8:00 p. m.

San Antonio Ophthalmo-Oto-Laryngological Society

President: Dr. James P. Aderhold, Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. Virgil S. Steele, South Texas Bldg., San Antonio, Texas.
Place: San Antonio Texas, and Brooke General Hospital. Time: 7 p. m., second Tuesday of each month from September to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. C. B. Cowan, 490 Post St., San Francisco.
Secretary: Dr. D. Harrington, 384 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. David C. Swearingen, Slattery Bldg., Shreveport, La.
Secretary-Treasurer: Dr. Kenneth Jones, Medical Arts Bldg., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President. Dr. Robert L. Pohl, W. 1104, 21st Ave., Spokane, Wash.
Secretary: Dr. Malcolm N. Wilmes, 407 Riverside Ave., Spokane, Wash.
Place: Spokane Medical Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. A. H. Rubenstein, 713 E. Genesee St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. H. Blaisdell, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toledo Eye, Ear, Nose and Throat Society

Chairman: Dr. W. W. Randolph, 1838 Parkwood Ave., Toledo 2, Ohio.
Secretary: Dr. John L. Roberts, 316 Michigan St., Toledo, Ohio.
Place: Toledo Club. Time: Each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. R. G. C. Kelly, 14 Lynwood Ave., Toronto 5, Canada.
Secretary: Dr. Alfred J. Elliot, 802 Medical Arts Bldg., Toronto 5, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. Richard W. Wilkinson, 1408 L St. N. W., Washington, D. C.
Secretary Treasurer: Dr. Jerome A. Sansoucy, 1710 Rhode Island Ave. N. W., D. C.
Place: Medical Society of District of Columbia Bldg., 1718 M St. N. W., Washington, D. C. Time: 7:30 p. m., first Monday in November, January, March and May.

Wilkes-Barre Ophthalmological Society

Chairman: Each member in turn.
Secretary: Dr. Samuel T. Buckman, 70 S. Franklin St., Wilkes-Barre, Pa.
Place: Office of chairman. Time: Last Tuesday of each month from October to May.